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RESPIRATORY CARE

A MONTHLY SCIENCE JOURNAL
35TH YEAR—ESTABLISHED 1956

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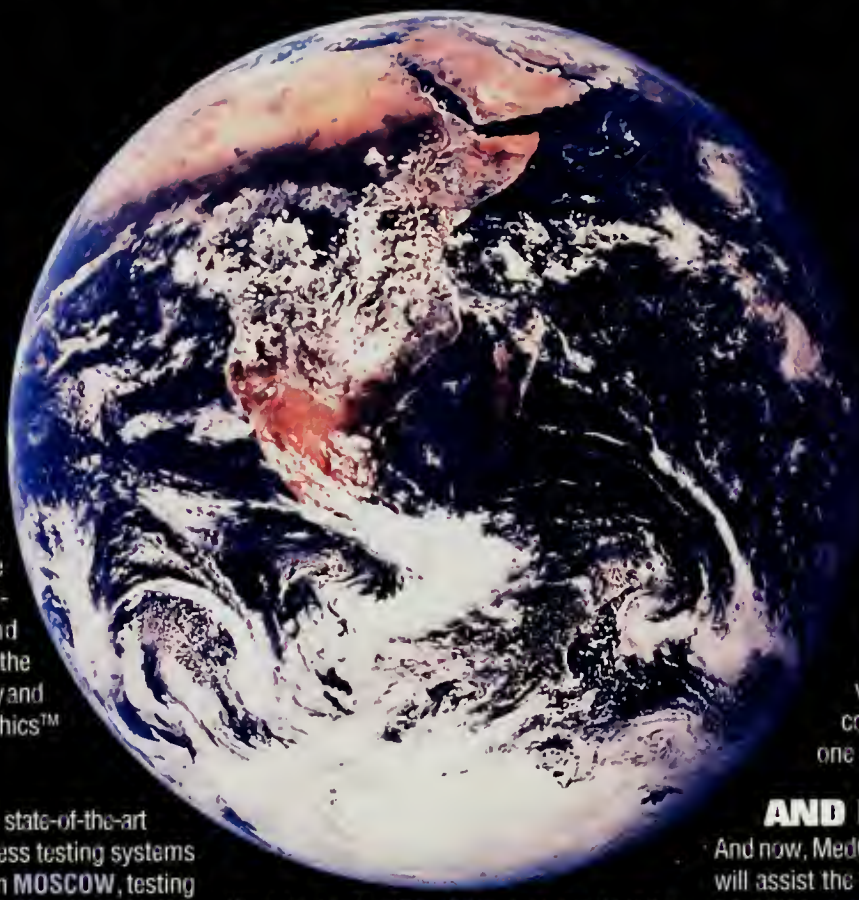

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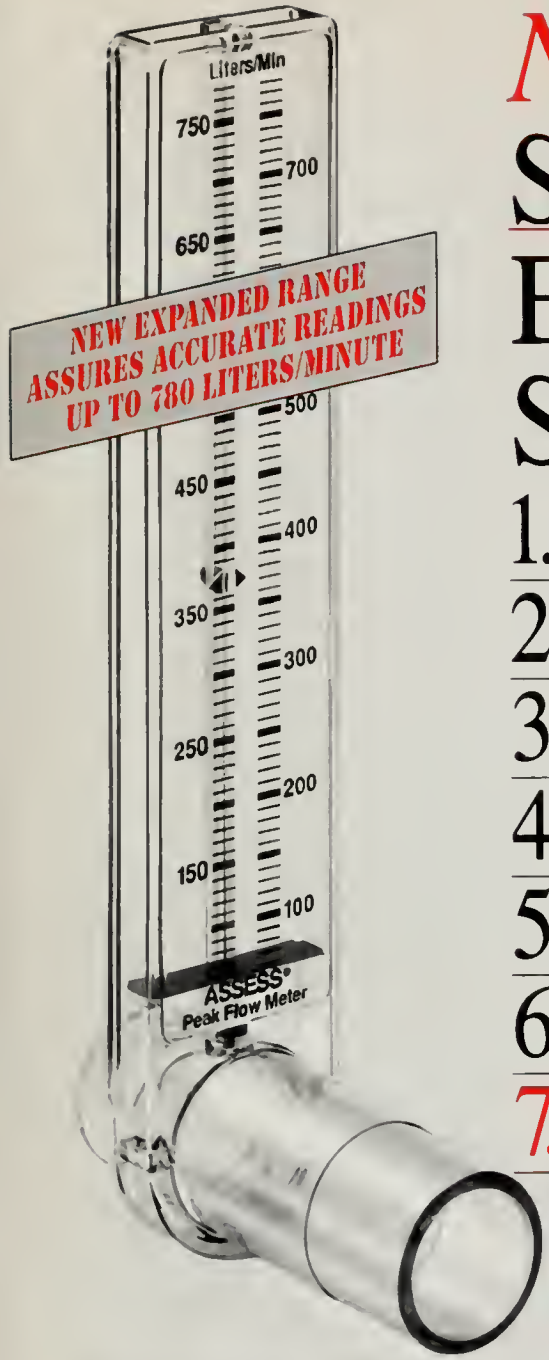
The following abstracts of pertinent articles that appeared in other journals during the years 1955 to 1957 reflect the mileu into which our journal was born.

Retrolental Fibroplasia in the United Kingdom: A Report to the Medical Research Council by Their Conference on Retrolental Fibroplasia—Br Med J 1955;2(July 9):78. (Reprinted from JAMA 1955;159:630.)

In October, 1951, when very little was known of the etiology and incidence of retrolental fibroplasia, a detailed investigation was set in train by the Medical Research Council. Premature baby units in hospitals spread throughout the United Kingdom were asked to complete a questionnaire relating to each mother during her pregnancy and to the treatment and development of the baby up to age of 6 months. Observations were recorded at 17 such units during the preiod October 1, 1951, to May 31, 1953. During the 20 months of the inquiry, 1,999 babies of 4 lb (1,800 gm) or under entered the nurseries, and the 1,095 of them who survived for at least two months form the basis of this report. Of these 1,095 babies, 84 (7.7%) showed evidence of a retinopathy, the criterion being the presence of neovascularization in the fundus, which was found during routine opthalmoscopy performed at regular intervals on all the children. In 39 of the 84 infants there was a subsequent regression, or return to normal fundi; in 45 there was blindness, implying one or both eyes permanently affected, with almost certain severe loss of vision. It was found that the cause of prematurity and maternal factors other than vaginal bleeding did not influence the subsequent development of the retinopathy. Boys were more often affected than girls; the main reason for this appears to be that they are more immature than girls for a given birth weight. The nurseries showed wide variation in their use of oxygen, and, in general, corresponding variations in the incidence of the disease. The use of oxygen predisposed to the development of a retinopathy. Except in two cases, at least five days' treatment with oxygen was required to produce the disease; and the likelihood of a baby getting the disease increased as the treatment with oxygen was prolonged. There are many other variables to be taken into account, but nurseries in which little oxygen was given and which were free from cases of progressive retinopathy did not, on the average, experience survival rates inferior to nurseries using more oxygen. Clinical signs of anoxia at and after birth were associated with a higher incidence of retinopathy, but on the average more oxygen was given to anoxic babies.

Chronic Bronchitis: The Effect of Cigarette Smoking—NC Oswald, VC Medvei. Lancet 1955;2(Oct 22):843. (Reprinted from JAMA 1955;159: 1794.)

The authors mention the following six factors as contributing to the development of chronic bronchitis: infection, polluted inspired air, the accompaniments of poverty, allergy, heredity, and the degenerative changes of advancing years. In addition, chronic bronchitis may be secondary to various pulmonary and general diseases. Assessment of the relative importance of any one factor is likely to be hazardous and can best be undertaken by selecting a group of people as similar as possible in all other respects. In the present instance the factor of cigarette smoking was investigated, and comparison was made of the prevalence of minor respiratory symptoms and of bronchitis in smokers and nonsmokers. The prevalence of minor respiratory symptoms and bronchitis was investigated in 5,844 clerical workers and was compared with their smoking habits. The proportion of clerical workers having minor respiratory symptoms and bronchitis was consistently higher in smokers than in nonsmokers. The greater prevalence of bronchitis in the older age group was clearly shown. For the entire series, 36% of the smokers were free from respiratory symptoms, compared with 53% of the nonsmokers. About 48% of the smokers and 38% of the nonsmokers had "mild respiratory symptoms." This difference must be accounted for to some extent by the so-called smoker's cough. The prevalence of bronchitis in smokers of the two sexes is very similar; the differences



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in nonsmokers are small. These findings suggest that sex is not an important factor in the development of bronchitis among persons living and working in similar circumstances.

The Use of Prednisone (Metricorten) in Respiratory Disease: II. Pulmonary Emphysema and Pulmonary Fibrosis—HA Bickerman, GJ Beck, AL Barach. J Chron Dis 1955;2(Sept): 247. (Reprinted from JAMA 1955; 159:1166.)

Results of administration of prednisone to patients with bronchial asthma were reported by the authors in a previous paper (JAMA 1955; 158(Aug 13):1396). Similar methods of administration of predinisone were used in 50 patients, 12 of whom had extensive pulmonary fibrosis complicated by varying degress of bronchopulonary infections; the remaining 38 patients had pulmonary emphysema, which was associated with fibrosis in 7. There were 39 male patients and 11 female patients, all between the ages of 15 and 74. The drug was given in four divided doses daily. The average daily dose of prednisone given was 50 mg on the first day, 35 mg for the first week, and 20 mg (maintenance dose) for 60 days. The results obtained differed from those obtained with cortisone and corticotropin (ACTH) in the following ways. Moderate to marked improvement with relief of dyspnea and increased tolerance occurred in 43 patients (86%) treated with a daily dose of prednisone averaging one-quarter that employed with cortisone. The onset of subjective improvement was significantly more rapid than with cortisone, usually within 48 hours after treatment was instituted. A striking loss of body weight ranging between 3 and 9 lb (1.3 and 4.1 kg) occurred in 12 of the 50 patients in the course of the first week of therapy, including patients with cor pulmonale who had not responded favorably to cortisone. A decrease of 8 and 9 lb (3.6 and 4.1 kg), respectively, occurred during the first week of treatment with prednisone in two of four patients who had not previously been treated with cortisone. The possible mechanism of this weight loss at a time when appetite and food intake were increased deserves further study. With the exception of salt and water retention and the decreased incidence of moon face or fat deposition, other untoward reactions observed with cortisone were observed likewise with prednisone, especially gastrointestinal disturbances and reactivation of sinobronchial infection. As a precautionary measure, administration of prednisone with milk and the addition of aluminum hydroxide or calcium carbonate two hours after meals and on retiring is advocated. Concurrent administration of the broad spectrum antibiotics, such as tetracycline, is suggested, particularly in patients with bronchitis or bronchiectasis.

The Pulmonary Hyaline Membrane Syndrome—SS Stevenson, LE Laufe. Pennsylvania Med J 1955;58(Oct): 1101. (Reprinted from JAMA 1955; 159:1491.)

The pulmonary hyaline membrane syndrome has shown a real and alarming increase in incidence in the last 15 years. It now seems to be responsible for half the total mortality among premature infants and for more neonatal deaths in all live-born infants than any other cause. The infant presenting this syndrome, usually born prematurely or by cesarean section or to a diabetic mother, has mild respiratory difficulty shortly after birth as if he had aspriated amniotic fluid. He then seems to be well for three or four hours, after which breathing becomes progressively more labored. Repeated roentgenograms show increasing diffuse haziness of the lung fields, and the infant becomes more and more cyanotic. Approximately 50% of such babies die, usually before they are 48 hours old. Microscopic examination of the lungs reveals extensive atelectatis, vascular congestion, and an eosinophilic hyaline-like membrane that lines occasional dilated alveoli and alveolar ducts. Experiments with guinea pigs have shown that, when amniotic fluid, which has a thromboplastic-like activity, is superimposed upon pulmonary edema, 20% of the animals exhibit the respiratory symptoms of the disease and present the complete syndrome at postmortem examination. It seems probable, therefore, that the human disease is produced when aspirated amniotic fluid becomes mixed with a pulmonary exudate. The formation of pulmonary exudate in human infants may be due to several causes, including irritation of the lungs by high concentrations of oxygen. This last may well be important, especially in premature infants. The increase in the incidence of the pulmonary membrane syndrome, which occurred simultaneously with that of retrolental fibroplasia, took place during the years in which incubators capable of maintaining high concentrations of oxygen became available. Further work on the origin of the syndrome should be directed toward elucidating the causes of pulmonary edema in the neonatal period. Certain practical suggestions for the prevention and treatment of the disease can, however, be made on the basis of present knowledge. 1. Place newborn infants in a prone position. They will not smother, and constant postural drainage will be established. 2. Aspirate the stomachs of infants born by cesarean section to free them from amniotic fluid and debris. 3. Restrict oxygen to the amount needed to preserve life and prevent cyanosis. The concentration of oxygen should never exceed 45%. 4. Observe all newborn infants for heart failure and proceed to rapid digitalization if failure appears. 5. Prophylactic antibiotic therapy

should be given to every newborn baby with respiratory difficulty, especially when it is accompanied by atelectasis. 6. Be prepared to withdraw air from the pleural cavity if pneumothorax develops and makes it necessary. 7. Withhold food and parenteral fluids from infants with this syndrome until convalescence becomes obvious (usually within 48 hours). Cortisone, estrogen, and the fibrin-lysing substances are potentially dangerous and should be avoided. Bronchoscopy, which cannot remove foreign substances far down in the bronchial tree and which is traumatizing in newborn infants, should not be attempted.

Successful Treatment of Cardiac Arrest Despite "Delayed" Cardiac Massage—E Bucherl, R Koch. Thoraxchirugie 1956;4(Oct):261. (Reprinted from JAMA 1957;163: 586.)

Ventricular fibrillation and cardiac arrest developed during cardiac catheterization in a 50-year-old man. The electrocardiogram corroborated the cardiac arrest. Thoracic and abdominal rhythmical compression was begun at once, and oral intubation and administration of oxygen under excess pressure was begun between 2 and 3 minutes after the arrest. The electrocardiogram still showed ventricular fibrillation during the 5th minute. To facilitate opening of the thoracic cavity, the patient had to be removed from the roentgen table, and 7 minutes had elapsed before the chest was opened and 8 minutes before cardiac massage was begun. Normal cardiac function was restored, and no impairment of the cerebral function resulted. The favorable outcome despite the delayed cardiac massage is ascribed to the fact that a minimal circulation with optimal oxygenation was maintained by rhythmical compression of thorax and abdomen and by the administration of oxygen under excess pressure.

Lung Biopsy in Diffuse Pulmonary Disease—PA Theodos, FF Allbritten, RL Breckenridge. Dis Chest 1955; 27(June):637. (Reprinted from JAMA 1955;159:150.)

Lung biopsy under direct visualization through a small incision was performed with the aid of local anesthesia and with the lung kept expanded by positive pressure oxygen given through a snugly fitting face mask in 50 patients with diffuse pulmonary lesions. In 27 patients, a specific diagnosis was established by histological and bacteriological study of the biopsy specimen. In 26 of the 27 patients, the preoperative diagnosis was confirmed by biopsy. Lung biopsy is of greatest value in diffuse lesions such as those seen in pneumoconiosis, sarcoidosis, metastatic malignancy, and tuberculosis, where a specific pathological entity is present. In these conditons the diagnosis can be readily established, since the abnormal findings are sufficiently well-defined to permit accurate diagnosis. No specific pathological diagnosis was possible in 23 patients. Biopsy served only to rule out the presence of a definite disease entity in these patients. Appreciation of the high prevalence of nonspecific fibrosis and emphysema in clinical practice should make the use of lung biopsy more selective. The procedure was well tolerated by the authors' patients and produced minimal complications, except in one patient who died of cardiac failure secondary to pulmonary insufficiency three days postoperatively. In general lung biopsy affords a rapid, accurate method for diagnosis, determination of the medicolegal status, and determination of prognosis of diffuse pulmonary lesions. The use of the procedure as a diagnostic aid shortens the period of hospitalization and lessens the cost.

The Physiologic Closure of the Ductus Arteriosus in the Newborn Infant—FL Eldridge, HN Hultgren. J Clin Invest 1955;34(July):987. (Reprinted from JAMA 1955;159: 732.)

The exact time of functional closure of the ductus arteriosus in the newborn human infant is still unsettled, but there is evidence that, at least in some infants, the ductus may be patent during the first few hours of life and that it may be the site of a venoarterial shunt of considerable magnitude. The present study was stimulated by the observation that infants in the neonatal period occasionally show cyanosis of the lower part of the body but not of the upper portion. Regional cyanosis of this type has been observed in older children and adults who have a patent ductus arteriosus with pulmonary hypertension and a venoarterial or reversed shunt through the ductus. Owing to this shunt of venous blood through the ductus to the descending aorta, the arterial oxygen saturation is lower in the feet than in the hands. The authors believed that, if a similar shunt were present in the newborn infant, it would continue as long as the ductus remained patent and the pressure in the pulmonary artery exceeded that in the aorta. The authors determined the oxygen saturations of blood samples obtained from the hand and foot of 60 infants from 40 minutes to 11 months after birth. It was found that, in a majority of infants under 3 hours of age, and in many infants under 3 days of age, the foot saturations were significantly lower than hand saturations. Hypoxia increased or reestablished the hand-foot oxygen saturation difference in infants up to 15 hours

of age who had minimal initial hand-foot differences. The following implications have been drawn from these studies. 1. The ductus arteriosus may be open in the newborn human infant. 2. Under these circumstances it is the site of a venoarterial shunt of considerable magnitude that persists in a significant number of infants up to 72 hours of life. 3. A high pulmonary artery pressure is present at birth and persists as long as the venoarterial ductal shunt exists. 4. The cessation of the venoarterial ductal shunt is probably due to a fall in pulmonary artery pressure below the pressure in the aorta, rather than to an actual closure of the ductus. 5. Studies of blood gases in newborn infants in which heel blood has been used are not representative of true arterial blood, since an admixture of venous blood from the ductus may be present. 6. Heart murmurs are rarely heard in the newborn infant, in spite of the early physiological patency of the ductus arteriosus.

Dangers and Management of Retained Tracheobronchial Secretions—HT Ransdell Jr. Am Surgeon 1955;21(Oct):1001. (Reprinted from JAMA 1955;159:1687.)

There are patients who literally drown in their own tracheobronchial secretions. Normally these secretions are eliminated by ciliary action and by cough. If anything interferes with this elimination, eg, impairment of the cough reflex or ciliary action, these secretions dam up with resulting obstruction of the airway, atelectasis, hypoxia, pneumonia, and death. Factors responsible for excessive tracheobronchial secretions and their retention are: aspiration of oral and pharyngeal secretions; chronic bronchitis (often secondary to heavy smoking) and mild bronchiectasis; inability of the patient to cough and expectorate; unconsciousness of patient as with narcotics, anesthetic agents, and coma or comatose states; ineffectual cough due to pain or tight abdominal binders; tenacious secretions; paradoxical motion of chest or mediastinum as with thoracoplasty, multiple rib fractures, and sucking wounds of the chest; semi-Fowler's position, which favors collection of secretions in the bases of the lungs; and atelectasis. The most important symptom of retained tracheobronchial secretions is a relatively unproductive, wet, "rattly" cough. With more advanced bronchial obstruction, dullness, diminished breath sounds, and fremitus usually are present. Rhonchi detectable by stethoscope usually are a late or pneumonic expression of retained secretions. Roentgenograms often are of great help and suggest retained secretions if the lung appear poorly aerated and if atelectasis is demonstrated. Patients should be trained to cough. During major surgery an endotracheal tube should be used to facilitate aspiration of secretions and, before the patient is removed from the table, all pharyngeal and tracheobronchial secretions should be removed. The patient is encouraged to cough at least every hour during his first 24 to 48 hours after operation and at gradually increasing intervals thereafter. His position is changed, side to back and back to side. As soon as possible, the patient should be gotten out of bed and encouraged to walk. The following aids facilitate coughing: several deep breaths or a little swallow of water just prior to coughing, support of the operative incision during the coughing either by the attendant or the patient himself, and having the patient sit up while coughing. Inhalation of high humidity atmosphere for 20 to 30 minutes, three or four times daily, is helpful in thinning or preventing drying of the secretions and thus making them easier to cough up. Ammonium chloride in 1 gm doses every two to four hours, is very helpful in this respect. Bronchoscopy and tracheobronchial catheter aspiration is necessary in patients unable to remove their tracheobronchial secretions. Hardly anything is more gratifying than to see a cyanotic moribund patient with the "death rattle" clear his cyanosis, reaerate his lungs, return to consciousness, and start on the road to recovery from an apparently hopeless situation, within 10 to 15 minutes after aspiration of his tracheobronchial secretions.

The Structure of the Respiratory (Terminal) Portion of the Lungs—BM Fried. Arch Int Med 1956; 98(Dec);691. (Reprinted from JAMA 1957;163:601.)

The lungs are the only visceral organ in contact with the outside world, but when removed from the chest under sterile conditions they contain no pathogenic bacteria. Yet a person inhales 12,000 liters of air in 24 hours, taking in enormous quantities of particulate matter and bacteria. When fine emulsions of pathogenic bacteria are injected into the air sacs by way of the trachea, they are retained and destroyed in the lungs without the occurrence of local disease. The author observed that a dose of pneumococci that kills a rabbit in approximately 3 days after injection into the blood stream is harmless to an animal when diplococci have been introduced into the pulmonary parenchyma by way of the trachea. The lungs possess a defensive mechanism made up of cells lying on and within the septums. These cells assume the functions of ameboid phagocytic macrophages. They also participate in the formation of the tubercle in tuberculosis and Boeck's sarcoid; they produce the elements that make up the silicious nodule in pneumonoconiosis; and they are the progenitors of the large foam cells in

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lipid pneumonitis and of the heart-failure cells in congestive heart failure. To all appearances, these cells are the elements commonly identified as respiratory epithelial cells lining the walls of the air sacs.

Artificial Hibernation in Neonatal Pathology—M Lacomme. Semaine hop 1954;30(Sept 10-14):3169. (Reprinted from JAMA 1955;157:86.)

A logical way of treating premature babies and arresting the pathological process (anoxia) operative in them that may lead to irreversible lesions is to put them in a state of hibernation, in order to lessen their oxygen requirements and maintain the integrity of their vascular systems. This can be accomplished with the use of ganglioplegics and cold. The main object of slowing down the babies' metabolic processes is to approximate their need of oxygen to their weak powers of absorption and distribution of it. Life at lowered temperature is possible because the body's defense reaction to cold is blocked by the ganglioplegic agents. The author's method is to use 1.5 mg of chlorpromazine (Largactil) (10-[γ-dimethylaminopropyl]-2-chlorophenothiazine hydrochloride) daily per kilogram of body weight in subcutaneous injection repeated every 40 minutes. Sometimes promethazine (Phenergan) is used in conjunction with the other drug. This treatment lasts not over three days, and usually from 24 to 48 hours. Vitamins K and P are given as antihemorrhagics, and neostigmine (Prostigmin) is given to combat the abdominal distension that comes with prolonged administration of ganglioplegics. The child is kept with no covering or clothing in an incubator at 28 °C (82.4 °F); his termperature stabilizes itself at 33 °C (91.4 °F). Rewarming is achieved by progressive diminution of the number and strength of the injections. Constant careful surveillance of each baby is essential to the technique. Artificial hibernation is indicated in babies who are in shock, who are premature, or who have had exchange transfusion. It was used by the author in 30 who were considered irretrievable; 7 survived. One wonders whether it would not be possible to obtain better results by long-continued hibernation. This does not seem feasible with ganglioplegics because their prolonged administration is harmful, but premature infants might be able to do without them, since, according to Ginglinger, they have no natural defense reactions to cold. This question is being studied at the present time by other French authors (Debray and Pierret, Breton, and Lefebvre), who place the babies in an incubator at 28 °C (82.4 °F) and let their temperatures regulate themselves spontaneously.

Tobacco as A Contributing Cause of Degenerative Coronary Disease—LH Sigler. New York J Med 1955; 55(Nov 1):3107. (Reprinted from JAMA 1955;159:1682.)

Of 1,186 men and 334 women with angina pectoris and coronary thrombosis, 519 men and 292 women were nonsmokers, while 667 men and 42 women had been habitual smokers since early youth. The possible effect of tobacco in expediting the development of degenerative coronary disease in these 1,520 patients, 985 of whom were living and 535 were dead, was studied by a comparison of the age at onset of the clinical manifestations of the disease, the age at onset of coronary occlusion, and the age at death from coronary disease in nonsmokers and smokers. Clinical manifestations of coronary disease before 50 years of age developed in only 29.2% of nonsmokers among men and in 19.1% of nonsmokers among women, as compared with as many as 44.8% of smokers among men and 50.2% among women. The higher the degree of smoking, the earlier the onset of the clinical manifestations of coronary disease. The first attack of coronary occlusion before 50 years of age occurred in only 27.7% of nonsmokers as compared with as many as 42.9% of smokers among men, and in only 14.7% of nonsmokers as compared with as many as 58.3% of smokers among women. The higher the degree of smoking in both men and women, the earlier the occurrence of the first attack of coronary occlusion. In male smokers as many as 20.2% died before 50 years of age, while in nonsmokers only 6.4% died before that age. In women as many as 46.5% smokers and 5.6% nonsmokers died before 50 years of age. In both men and women the higher the degree of smoking, the earlier the occurrence of death. Several possible mechanisms may be considered as the underlying factors in the earlier degeneration of the coronary vessels in smokers. One is the effect of nicotine on the sympathetic nerve ganglions inducing recurring angiospasm of the coronary vessels. Another possible cause may be a metabolic upset resulting from the effect of nicotine on various other parts of the body. A third factor might be a very low-grade prolonged anoxemia resulting from the carbon monoxide of tobacco smoke. A fourth factor may be an occasional allergy to tobacco. Although the earlier appearance of clinical manifestations of coronary disease and of coronary occlusion as well as the earlier death from such disease in smokers, observed in the author's series, would appear to suggest that tobacco expedites the development of coronary disease, one must consider the fact that the differences in the humber of smokers and nonsmokers in whom coronary disease developed were not marked. Tobacco may have some influence in expediting the development of coronary disease in a small proportion of the population,

but certainly it is not the sole cause of coronary disease, even in those patients in whom it appears to be operative. The underlying causes in all patients are probably heredity, metabolic disturbances, abnormalities in the chemical composition of the blood, and physical, mechanical, and other unknown factors.

Long-Term Chemotherapy in Chronic Bronchitis— JR May, NC Oswald. Lancet 1956;2(Oct 20):814. (Reprinted from JAMA 1957;163:79.)

Observations are presented in 37 patients, all of whom had advanced chronic bronchitis and attended the bronchitis clinic of a London hospital. The group included 36 men and 6 women, ranging in age from 36 to 72 years. All had active infection indicated by pus in the sputum. During 6 months of the winter of 1954-1955, 14 of the patients made daily observations on the volume and purulence of their sputum. Bacteriological examination of the sputum of these patients showed that *Hemophilus influenzae* was present persistently in every case. Treatment during these 6 months was entirely symptomatic, antibiotics being given only in short courses for acute exacerbations. For 6 months during the winter of 1955-1956 these 14 patients, together with the other 23 in the series, were given continuous chemotherapy. Because of the risk of causing blood dyscrasias, chloramphenicol was not used. Previous experience with chlortetracycline in bronchitis had not been encouraging, so the antibiotics used in the investigation were limited to oxytetracycline and tetracycline. The 14 patients observed during both winters are designated as the "controlled group." Four of the 14 patients could not tolerate a large enough dose of either drug, and 1 patient was unchanged at the end of 6 months' treatment with oxytetracy-

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cline. The responses of the remaining 9 patients were all judged to be good. With 1 exception the daily volume of the sputum was reduced, and in every case the pus was either eliminated or much diminished. The number of attacks of acute bronchitis was reduced in 7 patients. All 9 stated that the tablets had given them the best winter for years. Of the 23 patients without a control period, one had to stop treatment because of diarrhea and 7 others could take only minimal doses for the same reason, but in 3 of these the bronchitis improved, nevertheless. Ten of the 15 patients who had no side effects from the antibiotics were enthusiastic about the effect of the treatment on their general health. Their sputum decreased in volume and purulence, and they lost less time from work. The purulence of the sputum was reduced also in 4 of the other 5 patients, but none of the 5 were impressed with the treatment. In view of the fact that 22 of 37 patients benefited from the treatment, the authors conclude that this treatment seems to be valuable in preserving reasonable health in the later stages of purulent chronic bronchitis. About a quarter of the patients were carriers of antibiotic-resistant coagulase-positive staphylococci at the end of the trial.

Anoxia in the Acute Respiratory Infections of Childhood: Its Recognition and Treatment—B Morrison. Lancet 1955;2(Oct 8):737. (Reprinted from JAMA 1955;159:1693.)

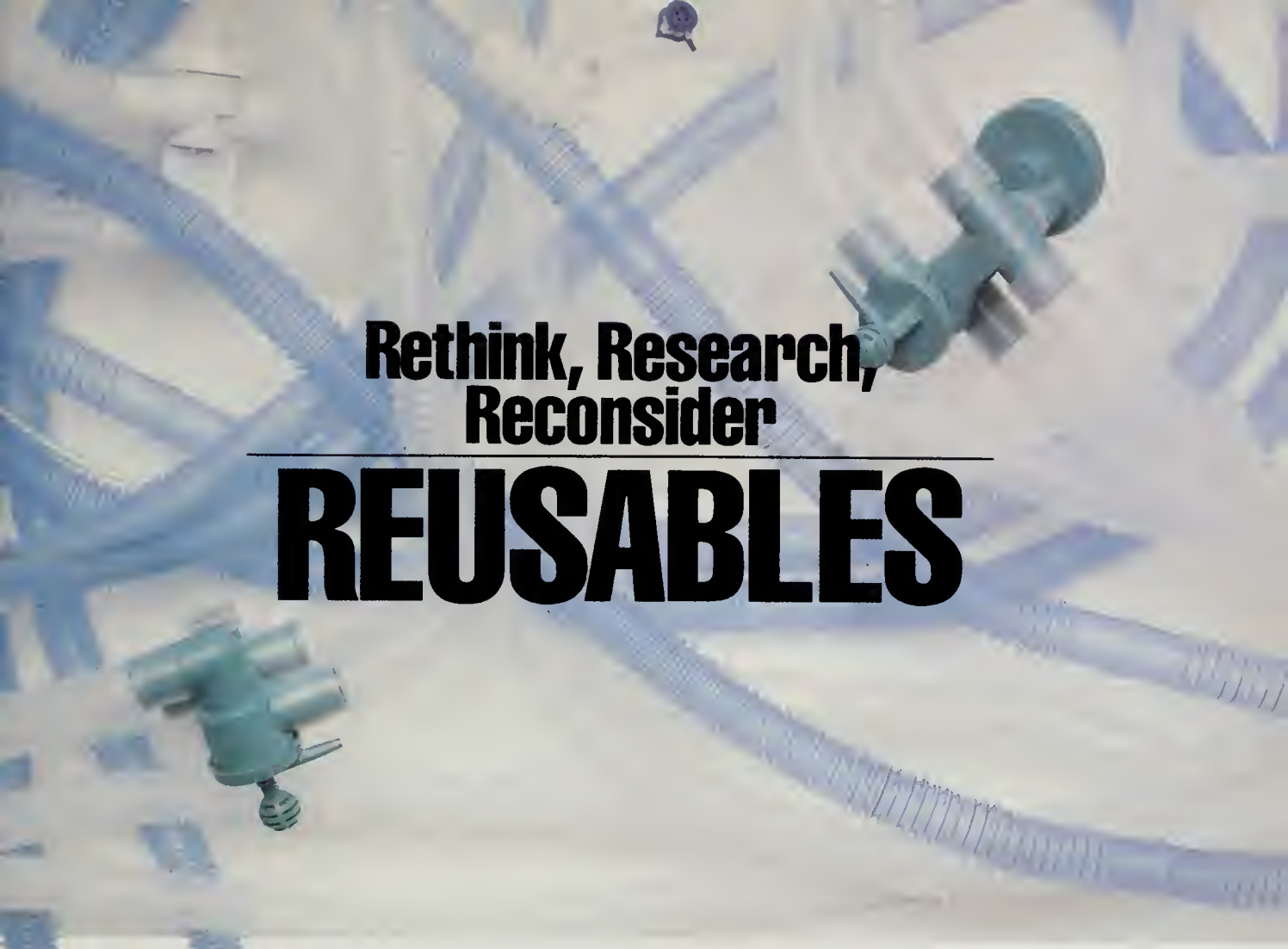
As the result of the control of bacterial infections by antibiotics, the clinical picture of acute respiratory infections in infants and children has undergone a considerable change. The toxic form of illness with hyperpyrexia and repeated convulsions that in the past often had a fatal outcome is now comparatively rare; however, anoxia has become more prominent and is now the main cause of death, largely because bronchiolitis, which is becoming increasingly common in infants under the age of one year, does not respond to antibiotic therapy and often produces a suffocative picture. The author studied the problem of anoxia as part of an investigation of severe respiratory infections in 108 infants and children. This report presents the results of correlating oxygen saturation estimations, clinical data, and response to oxygen therapy. It was found that direct estimations of arterial oxygen saturation are difficult and time-consuming, and as a method of assessing the need for and controlling oxygen therapy they are impracticable in routine hospital work. In this study, the author demonstrates the close relation between restlessness and anoxia and also the fallibility of relying on the child's color, at least in the lesser degrees of anoxia. The behavior of the infant emerged as the most reliable clinical guide in the control of oxygen therapy. Before making a diagnosis of anoxia, it is necessary to exclude restlessness due to hunger, discomfort, loneliness, or pain. In the presence of obvious respiratory distress, restlessness is usually due to anoxia. In some of the children, the standard oxygen tent, giving an oxygen concentration of from 40 to 45%, was inadequate for the relief of anoxia, and higher concentrations were required. These were supplied by a small tent, facemask, or special oxygen box designed to give up to 90% oxygen. With the use of these higher concentrations when necessary, the oxygen lack was relieved in 31 out of 32 children.

Antifoam Agents in Pulmonary Edema—RC Balagot, RM Reyes, MS Sadove. JAMA 1957;163:630.

The presence of frothy edema fluid in the alveoli of the lung hinders the diffusion of oxygen upon which the cells of the respiratory epithelium normally depend for their own oxygenation. This sets up a vicious circle, because the anoxic epithelium becomes more permeable and so permits further passage of fluid. The possibility of breaking this vicious circle by using antifoam agents was studied in rabbits after inducing pulmonary edema by intravenously injected epinephrine. Various antifoam agents were administered in a special chamber containing the experimental animals. Of the six agents tried, the most effective was a mixture containing a silicone and a polyhydric alcohol; it compared favorably with 95% ethyl alcohol in animals. It was therefore administered to eight patients with pulmonary edema by inhalation, with the aid of a respiratory apparatus affording intermittent positive pressure.

Clinical Study of Pneumoconiosis of Coal Workers in Ohio River Valley—MD Levine, MB Hunter. JAMA 1957;163:1.

Roentgenographic evidence of pneumoconiosis was found in 153 coal miners. Sixty of these were free from signs of cardiac disease other than cor pulmonale and returned for study often enough to permit satisfactory follow-up study. Complaints were referable to the chest in 32 cases; in the remaining 28 they chiefly involved other systems. The 60 with penumoconiosis were compared with a reference group of 74 miners matched as to age but free from roentgenographic signs of pneumoconiosis. The latter group ranged in age from 45 to 76 years, and the average time they had worked underground was 35 years. Physical and roentgenographic examinations were supplemented by laboratory tests. The men who remained free of roentgenographic abnormality showed a lower incidence of lower respiratory infection, emphysema, and dyspnea than did those with the roentgenographic lesions.



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Pneumonectomy, Lobectomy, and Segmental Resection in Treatment of Pulmonary Tuberculosis—EJ Zerbini, BJ Fleury de Oliveira, R Monteiro de Arruda. Rev paul med 1955;46(May):325. (Reprinted from JAMA 1955;159:224.)

Between 1946 and 1954, 255 patients with pulmonary tuberculosis were treated with one or more pulmonary resections. Males and females were about equal in number. Most of the patients were between 30 and 40 years of age. Bacilli were present in the sputum of all patients but 18. The patients were observed in three groups of 27, 76, and 152, as they had the operation during the first, second, or third three-year period of this study. Special reference is made to 152 patients who had the operation between 1951 and 1954. Indications for pulmonary resection were: unilateral diffuse tuberculosis (the "destroyed lung") in 14 cases; tuberculoma in 15; atelactasis, fibrosis, and bronchiectasis in 11; tuberculosis of either the lower or the middle lobe in 9; large cavities of upper lobe with bronchial tuberculosis in 17 cases; residual lesion after pneumothorax in 10 cases and after thoracoplasty in 15 cases; tuberculosis in only a lobe or in only a segment in 37 cases; and lesions of the upper lobe associated with lesions of either the lower or the middle lobe in 7 cases. Predominantly exudative tuberculosis was not observed in this group. There were 35 patients subjected to a pneumonectomy; 68 to a lobectomy; 38 to segmental resection; and 11 to a lobectomy combined with a segmental resection. Postoperative complications did not develop in 108 patients. Postoperative diffusion of tuberculosis developed in 10 patients, reactivation of the lesions in 2, bronchial fistula in 23, and empyema in 9. Other complications in nine patients were controlled by antibiotics or by collapse therapy. Clinical cure was obtained in 30 patients after segmental resection (78%), in 25 after pneumonectomy (71%) and in six after a lobectomy combined with a segmental resection (54%). There were eight deaths. In six patients only was death attributable to the operation. Five patients died after a pneumonectomy and one after a lobectomy, rate of mortality of 14% and 1% respectively for these operations. There were no deaths after segmental resection alone or in combination with a lobectomy.

The Management of Spontaneous Pneumothorax—AG Marrangoni, CF Storey, PO Geib. Am Rev Tuberc 1955;72(Sept):257. (Reprinted from JAMA 1955;159:1062.)

Spontaneous pneumothorax occurred in 80 otherwise healthy persons between 19 and 48 years of age who were admitted to the U.S. Naval Hospital in Portsmouth, Virginia. Spontaneous pneumothorax should not be considered a completely benign condition, because deaths resulting from it and complications associated with it have been reported. Thoracoscopy as an aid in diagnosis and in selecting the treatment of choice is emphasized. The rupture of a subpleural bleb is the most common cause of idiopathic spontaneous pneumothorax. Of the 80 patients, 55 were treated conservatively, ie, 49 by bed rest, 4 by needle aspiration, and 2 by needle aspiration and simple underwater seal without active suction. The overall average time required for reexpansion in these 55 patients was 30 days and the average period of hospitalization was 43 days. The remaining 25 patients were given the following active therapy. A trochar and cannula were introduced into the pleural space with the aid of local infiltration anesthesia. After withdrawal of the trochar, a thorascope was introduced through the cannula. After careful inspection of the surface of the collapsed lung and after determination of the cause of the collapse, the thoracoscope was withdrawn and the visceral and parietal pleurae were thoroughly poudraged with sterile talc that was insufflated with a powder blower. A rubber catheter was then placed through the cannula into the pleural space, the cannula was withdrawn, and active underwater seal suction at a negative presure of 15 to 20 cm of water was maintained for 24 hours. The patient then was placed on simple underwater seal drainage for an additional 24 hours. Penicillin was given intramuscularly as long as the intercostal catheter was in place. Pleural symphysis was achieved by talc poudrage and active underwater seal suction and reexpansion occurred within a few minutes. The average period of hospitalization required by these 25 patients was three days. This method appreciably decreases the time required for reexpansion. The decreased period of disability among patients thus managed results in savings in money and manpower when compared with conservative methods of treatment. Recurrent pneumothorax occurred in 12 of the 55 patients treated conservatively, but there was not a single case of recurrent collapse among the 25 patients who were given active treatment with talc poudrage and underwater seal catheter suction. Open thoracotomy was performed in three patients because numerous large emphysematous blebs were observed in each of them; this method of treatment is reserved for patients with large emphysematous blebs compressing healthy lung tissue. This is determined by roentgenographic and thoracoscopic examination of the chest. After the authors' paper had been submitted for publication, 27 additional patients with spontaneous pneumothorax were treated by thoracoscopy, intrapleural talc poudrage, and active suction. Prompt reexpansion has been accomplished in all these patients. There have been no complications or sequelae associated with the procedure.

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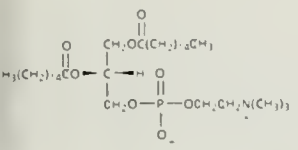
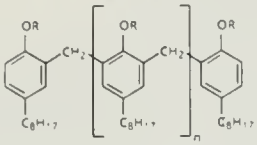


cetyl alcohol
(1-hexadecanol)
$CH_3(CH_2)_{14}CH_2OH$

tyloxapol
(formaldehyde polymer with oxirane and 4-(1,1,3,3-tetramethylbutyl)phenol)



[R is $CH_2CH_2O(CH_2CH_2O)_mCH_2CH_2OH$, m is 6 to 8, n is not more than 5]

NICAL PHARMACOLOGY: Surfactant deficiency is an important factor in the development of the neonatal respiratory tress syndrome (RDS) Thus, surfactant replacement therapy early in the course of RDS should ameliorate the disease l improve symptoms Natural surfactant, a combination of lipids and apoproteins, exhibits not only surface tension reducing perties (conferred by the lipids), but also rapid spreading and adsorption (conferred by the apoproteins). The major ction of the lipid component of natural surfactant is DPPC, which comprises up to 70% of natural surfactant by weight

hough DPPC reduces surface tension, DPPC alone is ineffective in RDS because DPPC spreads and adsorbs poorly In isurf Neonatal, which is protein free, cetyl alcohol acts as the spreading agent for the DPPC on the air-fluid interface oxapol, a polymeric long-chain repeating alcohol, is a nonionic surfactant which acts to disperse both DPPC and cetyl phol. Sodium chloride is added to adjust osmolality

rmacokinetics: Exosurf Neonatal is administered directly into the trachea Human pharmacokinetic studies of the ab-ption, biotransformation, and excretion of the components of Exosurf Neonatal have not been performed Nonclinical dies, however, have shown that DPPC can be absorbed from the alveolus into lung tissue where it can be catabolized ensively and reutilized for further phospholipid synthesis and secretion In the developing rabbit, 90% of alveolar sphospholipids are recycled. In premature rabbits, the alveolar half-life of intratracheally administered H^3-labeled phos-atidylcholine is approximately 12 hours

mal Studies: In animal models of RDS, treatment with Exosurf Neonatal significantly improved lung volume, compliance l gas exchange in premature rabbits and lambs The amount and distribution of lung water were not affected by Exosurf natal treatment of premature rabbit pups. The extent of lung injury in premature rabbit pups undergoing mechanical tilation was reduced significantly by Exosurf Neonatal treatment In premature lambs, neither systemic blood flow nor through the ductus arteriosus were affected by Exosurf Neonatal treatment Survival was significantly better in both mature rabbits and premature lambs treated with Exosurf Neonatal.

iical Studies: Exosurf Neonatal has been studied in the U S and Canada in controlled clinical trials involving more than 00 infants Over 10,000 infants have received Exosurf Neonatal through an open, uncontrolled, North American study signed to provide the drug to premature infants who might benefit and to obtain additional safety information (Exosurf natal Treatment IND).

ophylactic Treatment: The efficacy of a single dose of Exosurf Neonatal in prophylactic treatment of infants at risk of developing piratory distress syndrome (RDS) was examined in three double-blind, placebo-controlled studies, one involving 215 nts weighing 500 to 700 grams, one involving 385 infants weighing 700 to 1350 grams, and one involving 446 infants ghing 700 to 1100 grams The infants were intubated and placed on mechanical ventilation, and received 5 mL/kg surf Neonatal or placebo (air) within 30 minutes of birth

e efficacy of one versus three doses of Exosurf Neonatal in prophylactic treatment of infants at risk of developing RDS s examined in a double-blind, placebo-controlled study of 823 infants weighing 700 to 1100 grams. The infants were ubated and placed on mechanical ventilation, and received a first 5 mL/kg dose of Exosurf Neonatal within 30 minutes peat 5 mL/kg doses of Exosurf Neonatal or placebo (air) were given to all infants who remained on mechanical ventilation approximately 12 and 24 hours of age. An initial analysis of 716 infants is available

e major efficacy parameters from these studies are presented in Table 1

e 1 **Efficacy Assessments—Prophylactic Treatment**

Number of Doses Birth Weight Range	Single Dose 500 to 700 grams		Single Dose 700 to 1350 grams		Single Dose 700 to 1100 grams		One Versus Three Doses 700 to 1100 grams	
Treatment Group Number of Infants	Placebo (Air) N=106	EXOSURF N=109	Placebo (Air) N=185	EXOSURF N=176	Placebo (Air) N=222	EXOSURF N=224	One EXOSURF Dose N=356	Three EXOSURF Doses N=360
	% of Infants		% of Infants		% of Infants		% of Infants	
th ≤ Day 28[a]	53	50	11	6	21	15	16	9*
th through 1 Year[a]	59	60	14	11	30	20**	17	12*
th from RDS[b]	25	13*	4	3	10	5§	3	2
ct Cardiopulmonary Survival[a c]	29	25	69	76*	65	68	74	78
nchopulmonary Dysplasia (BPD)[a d] Incidence[b]	43 73	44 81	23 46	18 42	19 55	21 55	8 63	12 68

ntent-to-treat" analyses (as randomized) except for the 700 to 1350 gram single dose dy in which patients with congenital infections and anomalies were excluded s-treated" analyses

* p<0 05 ** p<0 01 § p=0 051

fined by survival through 28 days of life without bronchopulmonary dysplasia
fined by a combination of clinical and radiographic criteria

cue Treatment: The efficacy of Exosurf Neonatal in the rescue treatment of infants with RDS was examined in two double-blind, placebo-rolled studies One study enrolled 419 infants weighing 700 to 1350 grams, the second enrolled 1237 infants weighing 1250 grams and ve In the rescue treatment studies, infants received an initial dose (5 mL/kg) of Exosurf Neonatal or placebo (air) between 2 and 24 hours e followed by a second dose (5 mL/kg) approximately 12 hours later to infants who remained on mechanical ventilation. The major efficacy meters from these studies are presented in Table 2

e 2 **Efficacy Assessments—Rescue Treatment**

Number of Doses Birth Weight Range	Two Doses 700 to 1350 grams		Two Doses 1250 grams and above	
Treatment Group Number of Infants	Placebo (Air) N=213	EXOSURF N=206	Placebo (Air) N=623	EXOSURF N=614
	% of Infants		% of Infants	
th ≤ Day 28[a]	23	11***	7	4*
th through 1 Year[a]	27	15***	9	6§
th from RDS[b]	10	3**	3	1*
ct Cardiopulmonary Survival[a c]	62	75**	88	93**
nchopulmonary Dysplasia (BPD)[a d]	18	15	6	3*

ntent-to-treat" analyses (as randomized)
s-treated" analyses
fined by survival through 28 days of life without bronchopulmonary dysplasia
fined by a combination of clinical and radiographic criteria

* p<0.05 ** p<0 01 *** p<0 001 § p=0 067

Clinical Results: In these six controlled clinical studies, infants in the Exosurf Neonatal group showed significant improvements in FiO_2 and ventilator settings which persisted for at least 7 days Pulmonary air leaks were significantly reduced in each study Five of these studies also showed a significant reduction in death from RDS Further, overall mortality was reduced for all infants weighing >700 grams The one versus three-dose prophylactic treatment study in 700 to 1100 gram infants showed a further reduction in overall mortality with two additional doses

Safety information is presented in Tables 3 and 4 (see ADVERSE REACTIONS) Beneficial effects in the Exosurf Neonatal group were observed for some safety assessments Various forms of pulmonary air leak and use of pancuronium were reduced in infants receiving Exosurf Neonatal in all six studies

Follow-up data at one year adjusted age are available on 1094 of 2470 surviving infants Growth and development of infants who received Exosurf Neonatal in this sample were comparable to infants who received placebo

INDICATIONS AND USAGE: Exosurf Neonatal is indicated for:

1 **Prophylactic** treatment of infants with birth weights of less than 1350 grams who are at risk of developing RDS (see PRECAUTIONS),
2 **Prophylactic** treatment of infants with birth weights greater than 1350 grams who have evidence of pulmonary immaturity, and
3 **Rescue** treatment of infants who have developed RDS

For **prophylactic** treatment, the first dose of Exosurf Neonatal should be administered as soon as possible after birth (see DOSAGE AND ADMINISTRATION General Guidelines for Administration).

Infants considered as candidates for **rescue** treatment with Exosurf Neonatal should be on mechanical ventilation and have a diagnosis of RDS by both of the following criteria

1. Respiratory distress not attributable to causes other than RDS, based on clinical and laboratory assessments
2. Chest radiographic findings consistent with the diagnosis of RDS

During the clinical development of Exosurf Neonatal, all infants who received the drug were intubated and on mechanical ventilation For three-dose prophylactic treatment with Exosurf Neonatal, the first dose of drug was administered as soon as possible after birth and repeat doses were given at approximately 12 and 24 hours after birth if infants remained on mechanical ventilation at those times For rescue treatment, two doses were given, one between 2 and 24 hours of life, and a second approximately 12 hours later if infants remained on mechanical ventilation Infants who received rescue treatment with Exosurf Neonatal had a documented arterial to alveolar oxygen tension ratio (a/A) <0 22

CONTRAINDICATIONS: There are no known contraindications to treatment with Exosurf Neonatal.

WARNINGS:

Intratracheal Administration Only: Exosurf Neonatal should be administered only by instillation into the trachea (see DOSAGE AND ADMINISTRATION).

General:

The use of Exosurf Neonatal requires expert clinical care by experienced neonatologists and other clinicians who are accomplished at neonatal intubation and ventilatory management. Adequate personnel, facilities, equipment, and medications are required to optimize perinatal outcome in premature infants.

Instillation of Exosurf Neonatal should be performed **only** by trained medical personnel experienced in airway and clinical management of unstable premature infants. Vigilant clinical attention should be given to all infants prior to, during, and after administration of Exosurf Neonatal.

Acute Effects: Exosurf Neonatal can rapidly affect oxygenation and lung compliance

Lung Compliance: If chest expansion improves substantially after dosing, peak ventilator inspiratory pressures should be reduced immediately, without waiting for confirmation of respiratory improvement by blood gas assessment. Failure to reduce inspiratory ventilator pressures rapidly in such instances can result in lung overdistention and fatal pulmonary air leak

Hyperoxia: If the infant becomes pink and transcutaneous oxygen saturation is in excess of 95%, FiO_2 should be reduced in small but repeated steps (until saturation is 90 to 95%) without waiting for confirmation of elevated arterial pO_2 by blood gas assessment Failure to reduce FiO_2 in such instances can result in hyperoxia

Hypocarbia: If arterial or transcutaneous CO_2 measurements are <30 torr, the ventilator rate should be reduced at once Failure to reduce ventilator rates in such instances can result in marked hypocarbia, which is known to reduce brain blood flow

Pulmonary Hemorrhage: In the single study conducted in infants weighing <700 grams at birth, the incidence of pulmonary hemorrhage (10% vs 2% in the placebo group) was significantly increased in the Exosurf Neonatal group. None of the five studies involving infants with birth weights >700 grams showed a significant increase in pulmonary hemorrhage in the Exosurf Neonatal group. In a cross-study analysis of these five studies, pulmonary hemorrhage was reported for 1% (14/1420) of infants in the placebo group and 2% (27/1411) of infants in the Exosurf Neonatal group Fatal pulmonary hemorrhage occurred in three infants, two in the Exosurf Neonatal group and one in the placebo group Mortality from all causes among infants who developed pulmonary hemorrhage was 43% in the placebo group and 37% in the Exosurf Neonatal group

Pulmonary hemorrhage in both Exosurf Neonatal and placebo infants was more frequent in infants who were younger, smaller, male, or who had a patent ductus arteriosus Pulmonary hemorrhage typically occurred in the first 2 days of life in both treatment groups

In more than 7700 infants in the open, uncontrolled study, pulmonary hemorrhage was reported in 4%, but fatal pulmonary hemorrhage was reported rarely (0 4%).

In the controlled clinical studies, Exosurf Neonatal treated infants who received steroids more than 24 hours prior to delivery or indomethacin postnatally had a lower rate of pulmonary hemorrhage than other Exosurf Neonatal treated infants. Attention should be paid to early and aggressive diagnosis and treatment (unless contraindicated) of patent ductus arteriosus during the first 2 days of life (while the ductus arteriosus is often clinically silent). Other potentially protective measures include attempting to decrease FiO_2 preferentially over ventilator pressures during the first 24 to 48 hours after dosing, and attempting to decrease PEEP minimally for at least 48 hours after dosing

Mucous Plugs: Infants whose ventilation becomes markedly impaired during or shortly after dosing may have mucous plugging of the endotracheal tube, particularly if pulmonary secretions were prominent prior to drug administration. Suctioning of all infants prior to dosing may lessen the chance of mucous plugs obstructing the endotracheal tube If endotracheal tube obstruction from such plugs is suspected, and suctioning is unsuccessful in removing the obstruction, the blocked endotracheal tube should be replaced immediately

PRECAUTIONS:

General: In the controlled clinical studies, infants known prenatally or postnatally to have major congenital anomalies, or who were suspected of having congenital infection, were excluded from entry However, these disorders cannot be recognized early in life in all cases, and a few infants with these conditions were entered The benefits of Exosurf Neonatal in the affected infants who received drug appeared to be similar to the benefits observed in infants without anomalies or occult infection

Prophylactic Treatment-Infants <700 Grams: In infants weighing 500 to 700 grams, a single prophylactic dose of Exosurf Neonatal significantly improved FiO_2 and ventilator settings, reduced pneumothorax, and reduced death from RDS, but increased pulmonary hemorrhage (see WARNINGS) Overall mortality did not differ significantly between the placebo and Exosurf Neonatal groups (see Table 1) Data on multiple doses in infants in this weight class are not yet available Accordingly, clinicians should carefully evaluate the potential risks and benefits of Exosurf Neonatal administration in these infants

Rescue Treatment-Number of Doses: A small number of infants with RDS have received more than two doses of Exosurf Neonatal as rescue treatment Definitive data on the safety and efficacy of these additional doses are not available

Carcinogenesis, Mutagenesis, Impairment of Fertility: Exosurf Neonatal at concentrations up to 10,000 μg/plate was not mutagenic in the Ames Salmonella assay

Long-term studies have not been performed in animals to evaluate the carcinogenic potential of Exosurf Neonatal.

The effects of Exosurf Neonatal on fertility have not been studied

ADVERSE REACTIONS:

General: Premature birth is associated with a high incidence of morbidity and mortality Despite significant reductions in overall mortality associated with Exosurf Neonatal, some infants who received Exosurf Neonatal developed severe complications and either survived with permanent handicaps or died

In controlled clinical studies evaluating the safety and efficacy of Exosurf Neonatal, numerous safety assessments were made In infants receiving Exosurf Neonatal, pulmonary hemorrhage, apnea and use of methylxanthines were increased A number of other adverse events were significantly reduced in the Exosurf Neonatal group, particularly various forms of pulmonary air leak and use of pancuronium (See CLINICAL PHARMACOLOGY Clinical Results.) Tables 3 and 4 summarize the results of the major safety evaluations from the controlled clinical studies.

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Table 3 **Safety Assessments[a]—Prophylactic Treatment**

Number of Doses / Birth Weight Range	Single Dose 500 to 700 grams		Single Dose 700 to 1350 grams		Single Dose 700 to 1100 grams		One Versus Three Doses 700 to 1100 grams	
Treatment Group / Number of Infants	Placebo (Air) N=108	EXOSURF N=107	Placebo (Air) N=193	EXOSURF N=192	Placebo (Air) N=222	EXOSURF N=224	One EXOSURF Dose N=356	Three EXOSURF Doses N=360
	% of Infants		% of Infants		% of Infants		% of Infants	
Intraventricular Hemorrhage (IVH)								
Overall	51	57	31	27	36	36	38	35
Severe IVH	26	25	10	8	13	14	9	9
Pulmonary Air Leak (PAL)								
Overall	52	48	16	11	32	25	29	27
Pneumothorax	23	10*	5	6	19	11*	14	12
Pneumopericardium	1	4	2	0	<1	1	1	1
Pneumomediastinum	2	1	2	3	7	1**	3	2
Pulmonary Interstitial Emphysema	43	44	13	7*	26	20	23	22
Death from PAL	4	6	<1	<1	2	1	2	1
Patent Ductus Arteriosus	49	53	66	70	50	55	59	57
Necrotizing Enterocolitis	2	4	11	13	3	4	6	2*
Pulmonary Hemorrhage	2	10**	2	4	1	4	4	6
Congenital Pneumonia	4	4	2	4	2	2	1	1
Nosocomial Pneumonia	10	10	2	4	4	7	14	15
Non-Pulmonary Infections	33	35	34	39	28	29	35	34
Sepsis	30	34	30	34	23	24	30	27
Death From Sepsis	4	4	3	3	1	2	3	2
Meningitis	4	6	3	1	2	3	1	2
Other Infections	7	4	5	3	6	10	10	11
Major Anomalies	3	1	2	4	7	4	4	4
Hypotension	70	77	52	47	59	62	54	50
Hyperbilirubinemia	22	21	63	61	27	31	20	21
Exchange Transfusion	4	3	1	2	2	2	3	1
Thrombocytopenia[b]	21	25	not available		9	8	12	10
Persistent Fetal Circulation	0	1	1	1	0	2*	1	<1
Seizures	11	8	2	2	11	9	6	5
Apnea	34	33	76	73	55	65*	62	68
Drug Therapy								
Antibiotics	96	99	98	96	98	99	>99	99
Diuretics	55	60	39	37	59	63	64	65
Anticonvulsants	14	18	23	24	20	16	9	8
Inotropes	46	40	20	20	26	20	28	27
Sedatives	62	71	65	64	63	57	52	52
Pancuronium	19	11	22	14*	19	13*	15	11
Methylxanthines	38	43	77	77	61	72*	75	82*

[a] All parameters were examined with "as-treated" analyses
[b] Thrombocytopenia requiring platelet transfusion

* p<0.05
** p<0.01

Table 4 **Safety Assessments[a]—Rescue Treatment**

Number of Doses: / Birth Weight Range	Two Doses 700 to 1350 grams		Two Doses 1250 grams and above	
Treatment Group: / Number of Infants:	Placebo (Air) N=213	EXOSURF N=206	Placebo (Air) N=622	EXOSURF N=615
	% of Infants		% of Infants	
Intraventricular Hemorrhage (IVH)				
Overall	48	52	23	18*
Severe IVH	13	9	5	4
Pulmonary Air Leak (PAL)				
Overall	54	34***	30	18***
Pneumothorax	29	20*	20	10***
Pneumopericardium	4	1	1	2
Pneumomediastinum	8	4	5	2**
Pulmonary Interstitial Emphysema	48	25***	24	13***
Death from PAL	7	3	<1	1
Patent Ductus Arteriosus	66	57	54	45*
Necrotizing Enterocolitis	3	3	1	2
Pulmonary Hemorrhage	3	1	<1	1
Congenital Pneumonia	2	3	2	2
Nosocomial Pneumonia	5	7	2	2
Non-Pulmonary Infections	19	22	13	13
Sepsis	15	17	8	8
Death From Sepsis	<1	<1	1	<1
Meningitis	1	<1	1	<1*
Other Infections	5	8	5	6
Major Anomalies	3	3	4	4
Hypotension	62	57	50	39**
Hyperbilirubinemia	17	19	12	10
Exchange Transfusion	3	4	1	2
Thrombocytopenia[b]	10	11	4	<1**
Persistent Fetal Circulation	1	1	6	2**
Seizures	10	10	6	3*
Apnea	48	65**	37	44*
Drug Therapy				
Antibiotics	100	99	98	98
Diuretics	60	65	45	34***
Anticonvulsants	17	17	10	5**
Inotropes	36	31	27	16***
Sedatives	72	68	76	64***
Pancuronium	34	17**	33	15***
Methylxanthines	62	74**	49	53

[a] All parameters were examined with "as-treated" analyses.
[b] Thrombocytopenia requiring platelet transfusion

* p<0.05
** p<0.01
*** p<0.001

Pulmonary Hemorrhage: See WARNINGS

Abnormal Laboratory Values: Abnormal laboratory values are common in critically ill, mechanically ventilated, premature infants. A higher incidence of abnormal laboratory values in the Exosurf Neonatal group was not reported.

Events During Dosing: Data on events during dosing are available from more than 8800 infants in the open, uncontrolled clinical study (Table 5).

Table 5 **Events During Dosing in the Open, Uncontrolled Study[a]**

Treatment Type: / Number of Infants	Prophylactic Treatment N=1127	Rescue Treatment N=7711
	% of Infants	% of Infants
Reflux of Exosurf	20	31
Drop in O_2 saturation (≥ 20%)	6	22
Rise in O_2 saturation (≥ 10%)	5	6
Drop in transcutaneous pO_2 (≥ 20 mm Hg)	1	8
Rise in transcutaneous pO_2 (≥ 20 mm Hg)	2	5
Drop in transcutaneous pCO_2 (≥ 20 mm Hg)	<1	1
Rise in transcutaneous pCO_2 (≥ 20 mm Hg)	1	3
Bradycardia (<60 beats/min)	1	3
Tachycardia (>200 beats/min)	<1	<1
Gagging	1	5
Mucous Plugs	<1	<1

[a] Infants may have experienced more than one event.
Investigators were prohibited from adjusting FiO_2 and/or ventilator settings during dosing unless significant clinical deterioration occurred.

Reflux: Reflux of Exosurf Neonatal into the endotracheal tube during dosing has been observed and may be associated with rapid drug administration. If reflux occurs, drug administration should be halted and, if necessary, peak inspiratory pressure on the ventilator should be increased by 4 to 5 cm H_2O until the endotracheal tube clears.

>20% Drop in Transcutaneous Oxygen Saturation: If transcutaneous oxygen saturation declines during dosing, drug administration should be halted and, if necessary, peak inspiratory pressure on the ventilator should be increased by 4 to 5 cm H_2O for 1 to 2 minutes. In addition, increases of FiO_2 may be required for 1 to 2 minutes.

Mucous Plugs: See WARNINGS

OVERDOSAGE: There have been no reports of massive overdosage with Exosurf Neonatal.

DOSAGE AND ADMINISTRATION:

Preparation of Suspension: Exosurf Neonatal is best reconstituted immediately before use because it does not contain antibacterial preservatives. However, the reconstituted suspension is chemically and physically stable when stored at 2° to 30°C (36° to 86°F) for up to 12 hours following reconstitution.

Solutions containing buffers or preservatives should not be used for reconstitution. **Do Not Use Bacteriostatic Water for Injection, USP.** Each vial of Exosurf Neonatal should be reconstituted only with **8 mL** of the accompanying diluent (preservative-free Sterile Water for Injection) as follows: **1.** Fill a 10 mL or 12 mL syringe with 8 mL preservative-free Sterile Water for Injection using an 18 or 19 gauge needle. **2.** Allow the vacuum in the vial to draw the sterile water into the vial. **3.** Aspirate as much as possible of the 8 mL out of the vial into the syringe (while maintaining the vacuum), then SUDDENLY release the syringe plunger.

Step 3 should be repeated three or four times to assure adequate mixing of the vial contents. If vacuum is not present, the vial of Exosurf Neonatal should not be used.

The appropriate dosage volume for the entire dose (5 mL/kg) should then be drawn into the syringe from **below** the froth in the vial (again maintaining the vacuum). If the infant weighs less than 1600 grams, unused Exosurf Neonatal suspension will remain in the vial after the entire dose is drawn into the syringe. If the infant weighs more than 1600 grams, at least two vials will be required for each dose.

Reconstituted Exosurf Neonatal is a milky white suspension with a total volume of 8 mL per vial. Each mL of reconstituted Exosurf Neonatal contains 13.5 mg colfosceril palmitate, 1.5 mg cetyl alcohol, 1 mg tyloxapol, and sodium chloride to provide a 0.1 N concentration. If the suspension appears to separate, gently shake or swirl the vial to resuspend the preparation. The reconstituted product should be inspected visually for homogeneity immediately before administration. If persistent large flakes or particulates are present, the vial should not be used.

Dosage: Accurate determination of weight at birth is the key to accurate dosing.

Prophylactic Treatment: The first dose of Exosurf Neonatal should be administered as a single 5 mL/kg dose as soon as possible after birth. Second and third doses should be administered approximately 12 and 24 hours later to all infants who remain on mechanical ventilation at those times.

Rescue Treatment: Exosurf Neonatal should be administered in two 5 mL/kg doses. The initial dose should be administered as soon as possible after the diagnosis of RDS is confirmed. The second dose should be administered approximately 12 hours following the first dose, provided the infant remains on mechanical ventilation. A small number of infants with RDS have received more than two doses of Exosurf Neonatal as rescue treatment. Definitive data on the safety and efficacy of these additional doses are not available (see PRECAUTIONS).

Use of Special Endotracheal Tube Adapter: With each vial of Exosurf Neonatal Intratracheal Suspension, five different sized endotracheal tube adapters each with a special right angle Luer ®-lock sideport are supplied. The adapters are clean but not sterile. The adapters should be used as follows: **1.** Select an adapter size which corresponds to the inside diameter of the endotracheal tube. **2.** Insert the adapter into the endotracheal tube with a firm push-twist motion. **3.** Connect the breathing circuit wye to the adapter. **4.** Remove the cap from the sideport on the adapter. Attach the syringe containing drug to the sideport. **5.** After completion of dosing, remove the syringe and RECAP THE SIDEPORT.

Administration: The infant should be suctioned prior to administration of Exosurf Neonatal.

Exosurf Neonatal suspension is administered via the sideport on the special endotracheal tube adapter **WITHOUT INTERRUPTING MECHANICAL VENTILATION.**

Each Exosurf Neonatal dose is administered in two 2.5 mL/kg half-doses. Each half-dose is instilled slowly over 1 to 2 minutes (30 to 50 mechanical breaths) in small bursts timed with inspiration. After the first 2.5 mL/kg half-dose is administered in the midline position, the infant's head and torso are turned 45° to the **right** for 30 seconds while mechanical ventilation is continued. After the infant is returned to the midline position, the second 2.5 mL/kg half-dose is given in an identical fashion over another 1 to 2 minutes. The infant's head and torso are then turned 45° to the **left** for 30 seconds while mechanical ventilation is continued, and the infant is then turned back to the midline position. These maneuvers allow gravity to assist in the distribution of Exosurf Neonatal in the lungs.

During dosing, heart rate, color, chest expansion, facial expressions, the oximeter, and the endotracheal tube patency and position should be monitored. If heart rate slows, the infant becomes dusky or agitated, transcutaneous oxygen saturation falls more than 15%, or Exosurf Neonatal backs up in the endotracheal tube, dosing should be slowed or halted and, if necessary, the peak inspiratory pressure, ventilator rate, and/or FiO_2 turned up. On the other hand, rapid improvements in lung function may require immediate reductions in peak inspiratory pressure, ventilator rate, and/or FiO_2. (See WARNINGS and see below for additional information concerning administration.)

Suctioning should not be performed for two hours after Exosurf Neonatal is administered, except when dictated by clinical necessity.

General Guidelines for Administration: Administration of Exosurf Neonatal should not take precedence over clinical assessment and stabilization of critically ill infants.

Intubation: Prior to dosing with Exosurf Neonatal, it is important to ensure that the endotracheal tube tip is in the trachea and not in the esophagus or right or left mainstem bronchus. Brisk and symmetrical chest movement with each mechanical inspiration should be confirmed prior to dosing, as should equal breath sounds in the two axillae. In prophylactic treatment, dosing with Exosurf Neonatal need not be delayed for radiographic confirmation of the endotracheal tube tip position. In rescue treatment, bedside confirmation of endotracheal tube tip position is usually sufficient, if at least one chest radiograph subsequent to the last intubation confirmed proper position of the endotracheal tube tip. Some lung areas will remain undosed if the endotracheal tube tip is too low.

Monitoring: Continuous ECG and transcutaneous oxygen saturation monitoring during dosing are essential. In most infants treated prophylactically, it should be possible to initiate such monitoring prior to administration of the first dose of Exosurf Neonatal. For subsequent prophylactic and all rescue doses, arterial blood pressure monitoring during dosing is also highly desirable. After both prophylactic and rescue dosing, frequent arterial blood gas sampling is required to prevent post-dosing hyperoxia and hypocarbia (see WARNINGS).

Ventilatory Support During Dosing: The 5 mL/kg dosage volume may cause transient impairment of gas exchange by physical blockage of the airway, particularly in infants on low ventilator settings. As a result, infants may exhibit a drop in oxygen saturation during dosing, especially if they are on low ventilator settings prior to dosing. These transient effects are easily overcome by increasing peak inspiratory pressure on the ventilator by 4 to 5 cm H_2O for 1 to 2 minutes during dosing. FiO_2 can also be increased if necessary. In infants who are particularly fragile or reactive to external stimuli, increasing peak inspiratory pressure by 4 to 5 cm H_2O and/or FiO_2 20% just prior to dosing may minimize any transient deterioration in oxygenation. However, in virtually all cases it should be possible to return the infant to pre-dose settings within a very short time of dose completion.

Post-Dosing: At the end of dosing, position of the endotracheal tube should be confirmed by listening for equal breath sounds in the two axillae. Attention should be paid to chest expansion, color, transcutaneous saturation, and arterial blood gases. Some infants who receive Exosurf Neonatal and other surfactants respond with rapid improvements in pulmonary compliance, minute ventilation, and gas exchange (see WARNINGS). Constant bedside attention of an experienced clinician for at least 30 minutes after dosing is essential. Frequent blood gas sampling also is absolutely essential. Rapid changes in lung function require immediate changes in peak inspiratory pressure, ventilator rate, and/or FiO_2.

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The Effects on Respiration and Performance During Exercise of Adding Oxygen to Inspired Air RG Bannister and DJC Cunningham. J Physiol 1954;125(July):118. (Reprinted from JAMA 1955;157:96.)

Two atheletes and two other subjects ran on a motor-driven treadmill up various gradients. The intensity of the work was adjusted to ensure that each person reached his breaking point between the 7th and the 10th minute when he breathed atmospheric air. In other experiments he performed the same exercise while breathing 33, 66, or 100% oxygen. Addition of oxygen to the inspired air increased the time required to reach a breaking point. The performance was improved more by 66% and 100% than by 33% oxygen. With 66% oxygen three of the subjects did not reach a breaking point within 23 minutes. The discomfort that they had experienced when breathing air was replaced by a feeling of positive well-being. In contrast when breathing 100% oxygen they never felt elated, and all reached breaking points within 21 minutes. Oxygen reduced the pulmonary ventilation and the blood lactate response and allowed the alveolar carbon dioxide pressure to rise to higher levels. Two subjects exercised at a slight lower intensity of work. Sudden changes were made in the inspired gas mixtures from air to 66% or 33% oxygen in the course of the runs. These changes were followed rapidly by reductions in the pulmonary ventilation and increases in the alveolar carbon dioxide pressure. Subjective improvement occurred after a few breaths. On switching back to air, the reverse changes in the pulmonary ventilation and the alveolar carbon dioxide pressure followed rapidly. These effects were not observed during moderate exercise. Reasons are presented for regarding the respiratory effects of inhaling high concentrations of oxygen as being due to the abolition of an arterial anoxemia, which was thought to be present when air was breathed during exercise of more than a critical intensity. Relief of the anoxemia might exert its effects through the carotid and aortic chemoreceptors, or by improving cardiac function, or both. The authors do not consider it necessary to postulate the existence of an unknown respiratory stimulant liberated by muscles working under partially anaerobic conditions, though the possibility was not excluded. The existence of such a factor had been suggested by Asmussen and Nielsenin 1946. Discussing the depressant action of 100% compared with 66% oxygen, the authors suggest that it might be due to increases in the cerebral circulation, resulting from the excess of circulating carbon dioxide and lactate. Such an increase would nullify the protection from the deleterious effects of high-pressure oxygen afforded to the brain by the cerebral vasoconstriction that occurs at rest when pure oxygen is breathed.

The Pulmonary Manifestations of Fibroeystic Disease of the Pancreas PA di Sant'Agnese. Dis Chest 1955;27(June):654. (Reprinted from JAMA 1955;159:86.)

Between 1939 and 1954, cystic fibrosis of the pancreas, a generalized hereditary disease of children in which the pancreas, the lungs, the sweat glands, and frequently the liver are affected, was observed by the author in 291 children. Only 21 were hospitalized in the first five years, but 270 were admitted to the hospital in the last decade of the period studied. This increased incidence is a reflection both of greater recognition of the disease and of the advent of effective antibiotics, which greatly prolong the life span. The basic nature of the disease is not known, but it is probable that all exocrine glands are affected in this condition. A variable degree of bronchial and pulmonary involvement is a virtually constant feature of the disease, usually dominates the clinical findings, and determines the fate of the patient. Cystic fibrosis of the pancreas is the commonest cause of chronic nontuberculous pulmonary disease in the pediatric age group. Cystic fibrosis of the pancreas differs from other diseases of the lungs in that the basic phenomenon leading to respiratory involvement is a mechanical one, ie, failure to remove bronchial mucus, which perhaps is abnormal in its physicochemical characteristics. This results in widespread and severe bronchial obstruction and predisposes the patients to secondary infection. Bronchial obstruction may thus be considered as the primary and cardinal manifestation of the pulmonary component of fibrocystic disease of the pancreas. The causative agent is usually *Micrococcus (Staphylococcus) pyogenes var. aureus*. Of the 291 children with this diseae, 133 died. Of those still living, 29 were between the ages of 10 and 18 years; some of them were doing well. Early diagnosis and administration of antibiotics such as penicillin and streptomycin and of the broad spectrum antibiotics, such as oxytetracycline, chlortetracycline, and tetracycline, before the respiratory infection causes permanent damage to the bronchi, in addition to a high-protein, low-fat diet with adequate amounts of the liposoluble vitamins, added pancreatic extracts, and liberal amounts of salt in hot weather, offer the best hope of tiding these patients over into late childhood when the manifestations of this disorder seem to become milder.

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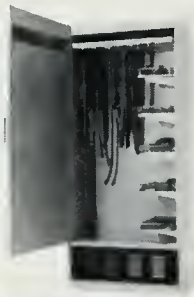

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Incidence of Clinical Hypersensitiveness of Medications in Patients with Bronchial Asthma—G Francheschini. Prog Med 1955;11(July 31): 431. (Reprinted from JAMA 1955; 159:1795.)

A study was made of 613 patients with bronchial asthma who were seen at the University of Rome outpatient clinic for allergic disease from 1952 to 1955 to determine the incidence and type of clinical hypersensitiveness to various medications. Skin reaction tests to exogenous allergens rsponsible for the asthmatic syndrome were positive in 310 patients, most of them in the 10- to 40-year age group. Of the 613 patients, 51 (8.38%) showed clinical hypersensitiveness to drugs—8.38% of these patients with a positive skin reaction and 8.24% with a negative skin reaction. Hypersensitiveness to acetylsalicylic acid was the most frequent (3.73%), and this high incidence is ascribed to the fact that acetylsalicylic acid is the medicament used most frequently by patients with bronchial asthma. Hypersensitiveness to aminopyrine and its derivatives was the next most common. The clinical manifestations of intolerance were urticaria, bronchial asthma, angioneurotic (Quincke's) edema, serum sickness, erythema, cutaneous purpura, and fever. In none of the patients were the drugs the first cause of asthma. They did, however, aggravate it, especially when it was already rather grave and had become resistant to the common therapies. Scratch tests with drugs were always negative except in one instance in which a patient showed a positive reaction to acetylsalicylic acid.

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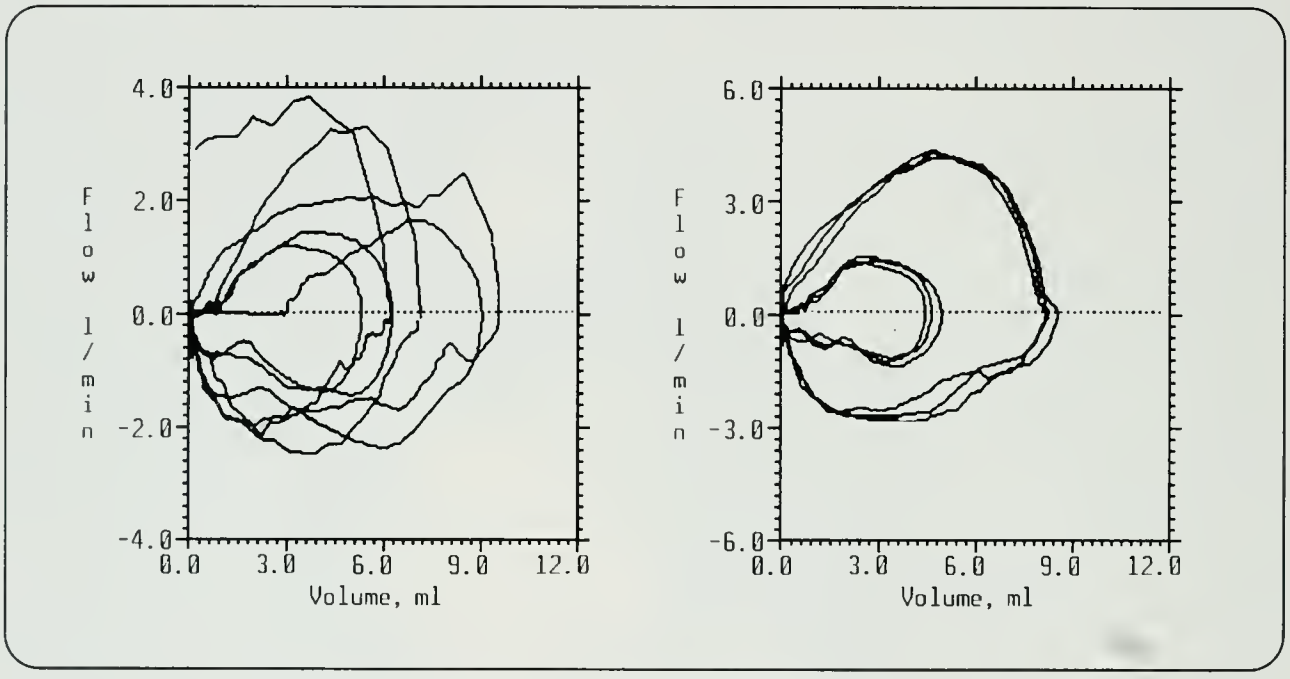

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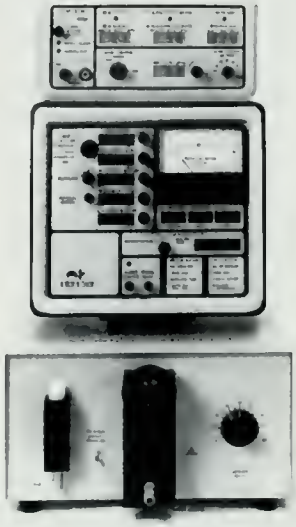

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A Clinical Study of Pulmonary Embolism GD DeLaughter Jr, WG Anlyan. Surg Gynecol Obstet 1956; 103(Dec):695. (Reprinted from JAMA 1957;163:393.)

During the past 6½ years the authors have seen in consultation all patients with venous thrombosis and pulmonary embolism on the surgical, gynecologic, and obstetric services of Duke Hospital, Durham, North Carolina. They report clinical observations on 60 patients with pulmonary embolism. Three of the emboli were fatal, two of them in the first episode. Thirty (50%) of the patients had emboli without any physical signs of peripheral venous thrombosis; it is assumed that these emboli arose from the pelvis. Of the 30 patients who had peripheral venous thrombosis (9 in the right, 11 in the left and 10 in both lower extremities) 20 had calf tenderness and 16 had leg pain, and Homan's sign was present in 12, leg swelling in 9, groin tenderness in 8, and pretibial vein dilation in 1. The diagnosis of pulmonary embolism is difficult and largely depends on the physician considering its possibility. If the patients present with acute pleuritic pain, dyspnea, hemoptysis, and cough, the diagnosis can be made if it is brought to mind. Often, however, the patient does not present this clear picture. Most patients initially have symptoms referable to the chest, and they are referred to as either a pain, a catch, a tight feeling, a fullness, an inability to take a deep breath, or a shortness of breath. A few hours later the patient may or may not produce blood-streaked sputum. These symptoms may be confused easily with myocardial infarction, angina, gallbladder disease, indigestion, or pneumonia. Examination frequently reveals an elevated pulse rate and localized rales. Roentgenograms may be helpful, but are seldom conclusive. Electrocardiograms have been useful in ruling out myocardial infarction. As soon as signs or symptoms suggest venous thrombosis or pulmonary embolism the patient should be treated with anticoagulant drugs, but clotting and prothrombin time should be ascertained prior to such treatment. Five patients had a pulmonary embolus while they were receiving anticoagulant therapy. Observations on these 5 patients caused the authors to modify their plan of treatment. They now rely on heparin in the early course of thromboembolism rather than on the coumarins. They also learned that pulmonary embolism after prostatic surgery should be be treated by inferior vena cava ligation if bleeding recurs with heparin therapy.

General Hypothermia in the Treatment of Massive Hemorrhage: Contribution to Study of Other Possibilities of Use in Transfusions of Blood and Substitutes —P Goffrini, E Bezzi. Presse méd 1955;63(June 1): 847. (Reprinted from JAMA 1955; 159:78.)

The authors summarize the results of their previous research, in which it was shown that dogs in hypothermia could survive and completely recover from massive, rapid exsanguination. The present series of experiments was performed on 22 dogs, 18 of which were exsanguinated under hypothermia and 4 at normal temperatures. Electrocardiograms, arterial pressures and respiration were studied to determine the exact mechanism of death from acute anemia. The two pathological factors, hemodynamic imbalance and anoxia, play distinct and separate parts. In the first period of hemorrhage, the changes caused by emptying of the heart and great vessels can be reversed and death prevented by proper fluid replacement; but in the second phase, characterized by progressive decrease in hemoglobin level and hematocrit, there appear disturbances of conduction, excitability, and contractitily of the myocardium that are due solely to anoxia. Four practical deductions are made from this work. 1. The clinical application of controlled hypothermia is of great value in the general management of patients in a state of acute hemorrhage, with respect to the consequences of anemic anoxia, though of course it is no substitute for transfusion therapy. 2. Although whole blood is the therapeutic agent of choice in acute hemorrhagic states, substitutes are useful in emergencies resulting from the use of controlled hypothermia. 3. In hemorrhagic states with particular contraindications to total reintegration of the circulating blood volume, it is possible to practice moderate application of a quantitative and qualitative replacement of the lost blood, since the body has a surprisingly high degree of tolerance to exsanguination. 4. In exchange transfusion alternate subtraction and substitution of blood in greater quantities is possible, with consequent simplification of technique and reduction of the inherent dangers of repeated insults to the circulatory equilibrium.

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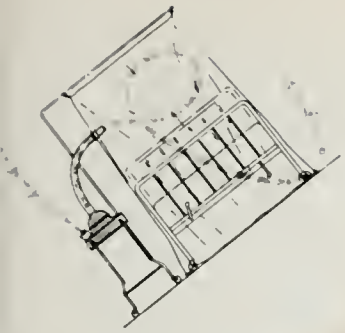

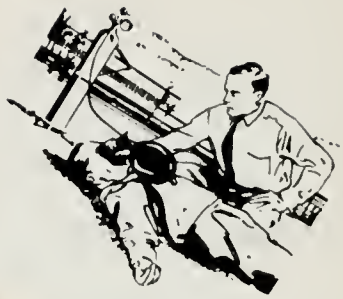

The Journal's 35th—Looking Back, Looking Ahead

Many factors contribute to the development of a profession—dedicated practitioners, appropriate educational advances, and improvements in technology being some of the most important. However, the single factor that best focuses attention on a profession's developments and achievements is the publication of research establishing the scientific basis of the profession. Our journal—RESPIRATORY CARE—is celebrating 35 years of documenting the development of our profession.

This commemorative issue presents selected articles from past volumes, each reflecting the scientific basis for the practice of respiratory care at the time of publication. Please note that these representative articles not only track the science of respiratory care, but also the development of the respiratory care professional. The ever expanding role of the respiratory care practitioner is well defined in the pages of our journal.

I encourage all respiratory care professionals to sit back and review our history, as documented in this issue, and to reflect on how far our profession has progressed over its short life. Consider also what the future holds for the profession of respiratory care and how each and every one of us can contribute to its continued development by documenting our scientific achievements in the pages of RESPIRATORY CARE.

Robert M Kacmarek PhD RRT
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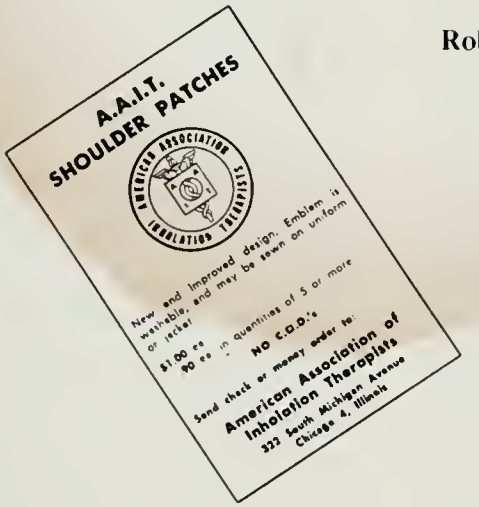
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Looking Over Our Shoulder: This Journal's First 35 Years

Happy birthday to the AARC journal RESPIRATORY CARE, which is 35 years old. The editors have chosen to mark the occasion by pausing to look back, reprinting in this issue some 21 papers that originally appeared in these pages from 1956 to 1986.

In looking over our editorial shoulder, we saw two themes that could be emphasized in this retrospective: The Expanding Role of the Respiratory Care Practitioner, which is represented by the reprinting of 10 papers; and Respiratory Care Devices and Techniques through the Years, revisited in 11 papers from the past. Before we get to those papers, though, shall we set the scene?

As It Was in the Beginning

Modern pulmonary medicine can arbitrarily be said to have begun about 1950, with publication of the document, "Standardization of Definitions and Symbols in Respiratory Physiology." (Dr Fred Helmholz, still active with the NBRC, was one of the authors.) The Engstrom ventilator appeared in 1952, just in time for the Copenhagen poliomyelitis epidemic. That early 'bible,' Comroe et al's book "The Lung," came along in 1955. Thus, this journal, beginning life in 1956, has coexisted with, and played a part in, nearly all the modern pulmonary medicine era.

The evolution of respiratory care is reflected in our professional organization's successive names: first the American Association for Inhalation Therapy (AAIT), then the American Association for Respiratory Therapy (AART), and now the American Association for Respiratory Care (AARC). This journal has had two names: INHALATION THERAPY, then RESPIRATORY CARE. With that second name, it led the way, introducing the broader term later adopted by the Association and, eventually, by many hospital services and schools.

1956 saw the AAIT journal INHALATION THERAPY start life under Editor James F Whitacre MS, in a world that would be unrecognizable to most of today's respiratory care practitioners. There was no credentialing system. Schools were almost nonexistent. The AAIT's 1955 national convention had drawn only 200 persons, many of them industrial representatives. In their hospitals, most early technicians had women bosses: inhalation therapy was usually under the nursing service (RNs), or central supply (RNs or LPNs), or anesthesia (CRNAs). Only about 1955 did anesthesiologists and chest physicians here and there begin to take on the inhalation therapy services, especially in the larger medical centers.

Founding Editor Jim Whitacre recalls that a typical inhalation therapy service covered only the day shift. "The other two shifts were covered by the orderlies on call, with the result that we had to spend half the morning ransacking the hospital to discover where they had taken our equipment." Several other features of the early days stick in Jim Whitacre's mind: "For a while, we had no departmental home, and our equipment was kept in Central Supply. There were no disposables then, so we had to wash everything, even tent canopies (what an operation!). The difficulty was that because we were shirttail cousins, regarded by CS as poaching on their territory, we could use the sinks only after they were through with them. A frustrating bottleneck.

"When we did get space of our own, it was situated in the basement at the back of the building as near the loading dock as possible, because we were only partly piped and had to use a lot of cylinder gas. The same dock and basement floor we had to traverse were also used by the commissary to bring in sides of beef. Blood was dripped all over the floor and often slipped in by one of us darting out the door on a stat call. Housekeeping used the same area to haul trash carts out to the incinerator, with various tidbits falling off the wagons as they passed by our doors.

"This was in a university medical center, mind you! It was years before we managed to cajole them into moving us up into the building where we were closer to the patients. We were not alone. It was common in those days for administrators very grudgingly to assign a small storage closet to the I.T. guy for his department. No plumbing, no electricity except for the ceiling light fixture. And oh, how they whined when we asked for work counters, outlets, and sinks. That row was topped only by the one they made quite a few years later when we asked for a desk, file cabinet, typewriter, secretary, and beepers. What did those things have to do with therapy? Were we trying to be the Administrator's office? Etc."

Other roles were peculiar to the time. Some inhalation therapy departments 'made' their own oxygen. In one small hospital in the early days, the new chief therapist found himself in trouble the second morning of his employment. An administrator chewed him out because the American flag in front of the hospital had not been lowered the evening before. It seemed that was the Inhalation Therapy Department's responsibility.

Licensure was mostly unheard of, except in New York City. As late as 1965, technicians preparing for the NYC licensure test had to bone up on BLB masks, even though nobody used them any longer. Why? Because the licensing inspector from the city health department was sure to ask a candidate about the use and care of these obsolete devices. There would be no questions about the Birds or Bennetts then in use, though—the licensure inspector didn't understand them. When the inspector wasn't testing inhalation therapy technicians, his job for the health department was to shoot rats on the waterfront docks. It was a different world.

And so the Journal was launched, with Jim Whitacre as Editor and Albert Carriere, the AAIT's Executive Director, as publisher. Given the lack of schools and the prior lack of formal communication among practitioners, the avowed purpose of INHALATION THERAPY in those early days was to educate its readers. Basic-information articles were contributed by educators employed by Linde, Ohio Chemical, National Cylinder Gas, Liquid Carbonic, and other equipment manufacturers or gas suppliers.

Again, Jim Whitacre recalls: "At the time, there were very few medical directors of inhalation therapy services, and writing articles for INHALATION THERAPY wasn't what they wanted to do, anyway. But our medical heroes like Ed Levine, Al Andrews, Vince Collins, Bill Miller, Duncan Holliday, Mike Saklad, Bob Lawrence, Fred Helmholz, and Hugh Mathewson transcribed their convention speeches for publication, wrote papers expressly for INHALATION THERAPY, or otherwise helped us. We did of course also have some therapist-written articles. We were lucky to have stalwarts like Vince Kracum and Don Gilbert who had directed departments long enough to have useful information to pass on. And there were a number of industrial/commercial people like Max Glasser, Abe Lister, and Larry Fruik, who got together articles for us on setting up home oxygen or equipment rental services.

"In a special category—nobody else like him—was Gary Gish (the only other therapist at that time with a Master's degree), who was active in a university research department in Chicago for a number of years before going to work for Puritan-Bennett, where his role as an educator of doctors, nurses, and therapists was, like Vince Kracum's, nothing short of stellar. Those two fellows wrote and spoke everywhere the length and breadth of the country for years, and hardly a therapist in the United States during the 50s, 60s, and 70s didn't know and respect them.

"Not surprisingly, there weren't many papers dealing with research. Few departments then were big enough to have any research activity. What papers were written inevitably went to medical journals. We reprinted many of the latter for our readers' education, but we strove mightily not to become a 'reprint journal.' At that stage of our evolution not more than a couple or so textbooks were available, so what our readers needed were informative articles about physiology, pathology, pharmacology, equipment developments, and therapy techniques. Therefore the nature of the contents of the Journal from year to year reflected the advances in the development of respiratory care."

If the early articles seem primitive, it should be understood that the articles in the major medical journals of the day were much the same—witness the abstracts from other journals reprinted in this issue. After all, modern medicine had begun only a few years before—let's say 1940, when penicillin was first produced in clinical quantities. So growing pains were in evidence. Respiratory care was advancing swiftly,

and INHALATION THERAPY reflected this, starting with a 5- by 7-inch format and four issues a year in 1956, then in 1960 growing to a 7- by 10-inch size and coming out six times a year.

Toward a Science Journal

In 1971, the name of the Journal changed to RESPIRATORY CARE. In 1973, the format grew up to the full 8½- by 11-inch size, and there were pictures on the covers. Growth remained rapid, and in 1974 RESPIRATORY CARE began to appear monthly. Research papers, case reports, and several new features reflected the vitality of the discipline. So many new industrial firms sprang up with the expanding technology that their product advertisements in the Journal almost overwhelmed the articles. A few readers complained.

After five years, the awful pictures disappeared from the covers. No longer a house organ for the Association, RESPIRATORY CARE was now clearly a scientific publication, with a mission to establish the scientific validity of the clinical and laboratory activities of respiratory care practitioners. The Open Forum became a Journal function. A strong Editorial Board set publication philosophy. Scientific conferences were sponsored, and their proceedings were published. The profession and the Journal had matured.

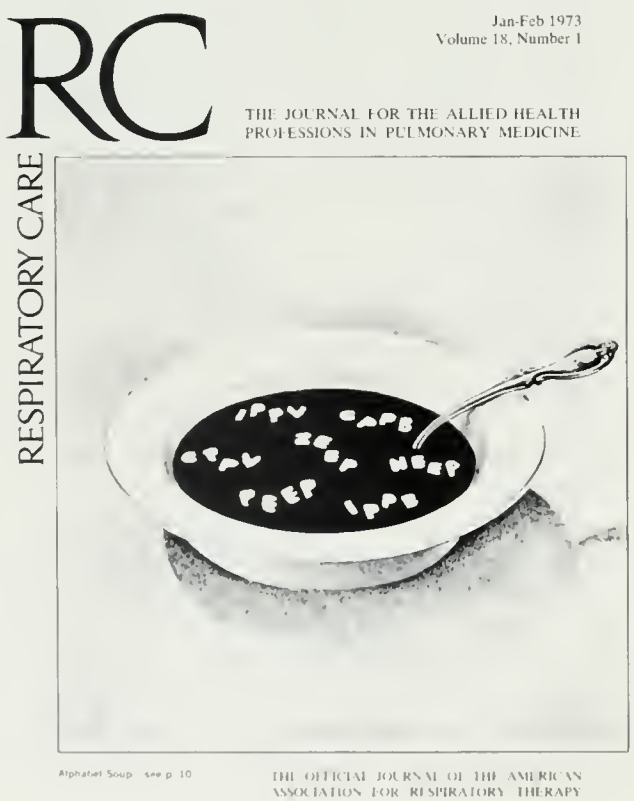
Today, everything indicates that RESPIRATORY CARE, like the discipline it serves, will continue to gain weight (a common phenomenon after age 35).

Entering the Twilight Zone

Where are we? What year is this? Oh, yes, it's December 1990, and we are returning, for this one Journal issue, to some of the scenes of yesteryear. First, let us look at The Expanding Role of the Respiratory Care Practitioner, evidenced in 10 articles from the past, and introduced by Jeri Eiserman. Then, a sampling of Respiratory Care Devices and Techniques through the Years is exemplified in 11 more articles, which Dean Hess introduces.

Happy birthday to you, RESPIRATORY CARE—and many more of them!

Phil Kittredge
Adjunct Editor
RESPIRATORY CARE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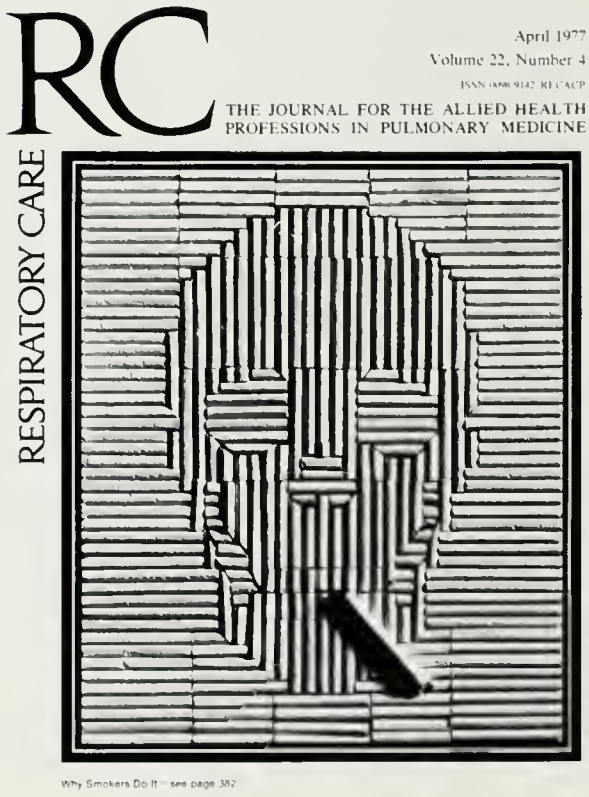
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The Expanding Role of the Respiratory Care Practitioner

Jeri E Eiserman MA RRT RCP

Since its inaugural issue in 1956, our Association's Journal has served a most important function by providing virtually the only written history of what could be described as the infancy and adolescence of an incredibly dynamic profession over the past 35 years. First under the name INHALATION THERAPY, later as RESPIRATORY CARE, past Journal issues provide us with a chronologic record of the significant events, ideas, opinions, and experiments that have shaped our growth and directed our path since the early years of our existence. When reading articles that have appeared in these pages during the past decades, one can detect the 'flavor' of our past and develop a new, or perhaps renewed, appreciation of where we are today.

It is my pleasure to provide some brief comments about 10 articles from the Journal's past volumes, which appear on the following pages, and which were chosen to help illustrate how the role of respiratory care practitioners has expanded. As I read through these articles, I was reminded that we, the practitioners of the 90s, stand on the shoulders of those who came before us. As you read these papers, you will also be reminded that we owe much to the physicians who, in the 50s and 60s, encouraged, trained, and trusted the pioneers in respiratory care. You will also note, as evidenced by the names of the authors, that this collaborative and collegial relationship continues today. I hope you will be intrigued and inspired by the work and dedication of the oxygen orderlies, inhalation therapists, and respiratory therapists who, through their efforts and spirit of adventure, provided the firm foundation upon which we now stand.

Ms Eiserman is Educational Consultant, Hudson RCI, Temecula, California.

The 10 papers that follow take us on a historical journey from early 1960 through August 1986. Each article was chosen because it illustrates—through topic, example, or editorial opinion—the expanding role of the respiratory care practitioner during that period. For the purpose of this 35th Anniversary issue, we use the term "role" to mean the function of the practitioner in direct relationship to patient care and health care in general—not to mean those roles internal to a respiratory care department or educational program, such as manager, supervisor, or instructor.

A Walk through History

During its first several years, INHALATION THERAPY functioned particularly as a source of basic education for its readers. After all, there were very few schools of inhalation therapy and only one or two textbooks around. At that time, the emphasis was on technical skills and the facility with which we dealt with therapeutic gases and equipment.

William C Allen focused on our role as technologic expert in his February 1960 article, **Therapists Must Become More Expert as Use of Respiratory Aids Grows**. He contended that inhalation therapists must increase their working knowledge of new equipment, particularly mechanical respirators and respiratory assisters, as therapists were often considered "the experts" in the use of this technology. Mr Allen went on to give an excellent overview of the devices of his day: tank and chest negative-pressure respirators, positive-pressure respirators (for IPPB), abdominal belts, and the rocking bed. If you haven't had an order to set up an abdominal belt or rocking bed lately, remember that this was 30 years ago.

A year later, in February 1961, Sister M Teresa asked INHALATION THERAPY's readers to look at the

inhalation therapist as something beyond an equipment wizard. In **Consider Not Only the Therapy But the Patient As Well**, she emphasized the importance of the therapist's words, deeds, and manner in the effective care of the patient. Sister Teresa underscored the importance of learning as much as possible about the patient, communicating in a knowledgable and comforting manner, and behaving with professionalism and competence. In this article we can see the role of the inhalation therapist going beyond an emphasis on technology to an emphasis on quality therapy. Unlike Mr Allen's 1960 paper, which has become outdated, Sister Teresa's message is still pertinent and valuable today.

In the April 1969 article by Ray Masferrer entitled **The Role of Patient Instruction in Improving IPPB Treatments**, we see evidence that the therapist's role was expanding to include the objective study and evaluation of treatment modalities and procedures. Mr Masferrer examined the importance of patient instruction by an inhalation therapist in effective IPPB, which was the major therapy of that time. The article illustrated the role that therapists could play in the scientific evaluation and documentation of inhalation therapy procedures.

The June 1969 issue was marked by a paper about respiratory home care—an early note on a theme that has become of major importance today. In **Home Care for Pulmonary Diseases**, Leone et al described a pioneering home care program for COPD patients, its objectives, and its outcomes. This article delineated the potential that home care held for the inhalation therapist as an avenue for expansion of his role outside the acute care hospital. In addition, the authors provided one of the first documented examples of the cost benefit of respiratory home care in the treatment of patients with chronic obstructive lung disease.

Two years later, in July 1971, Bryan and Andree documented the role of the respiratory therapist in performing endotracheal intubation. (Note the change in the practitioner's title. The Journal's name had changed the previous January—from INHALATION THERAPY to RESPIRATORY CARE.) In their paper, **Training Respiratory Therapists in Emergency Endotracheal Intubation**, the authors described a program implemented to train therapists in this skill. Not only did the article document the ability of trained therapists to successfully assume this role in a variety of clinical emergencies, but it also provided a blueprint for other hospitals seeking to set up such a program. Here we begin to see evidence of the respiratory therapist's role expanding into areas previously reserved for physicians or other medical specialists.

In October 1982, RESPIRATORY CARE published the results of a seminal study of the efficacy of home respiratory therapy. The article by Roselle and D'Amico, **The Effect of Home Respiratory Therapy on Hospital Readmission Rates of Patients with Chronic Obstructive Pulmonary Disease**, chronicled the work of the South Hills Home Health Agency in Homestead, Pennsylvania, a coal-mining region. The publication of this report displayed several important milestones.

First, the article emphasized the importance of the involvement of respiratory therapists in home care. Second, the United States Department of Health and Human Services, through association with the project, learned a great deal about respiratory care and the professionals rendering that care. Third, the article clearly documented that home care provided by respiratory therapists could significantly decrease the hospital readmission rates of COPD patients—thus legitimizing our role in the provision of respiratory care outside the acute care hospital. Fourth, the study underscored the role that therapists can play in the research necessary to document the scientific basis for what we do. And finally, the research reported in this paper led directly to a positive change in reimbursement policy for respiratory home care in Pennsylvania.

In February 1983 we see evidence that the therapist should expand his role beyond that of care provider. In their paper, **Review of a Bronchial Hygiene Evaluation Program**, Walton et al illustrated the positive impact that the respiratory therapist can have when he also becomes actively involved in patient assessment, care plan development, and outcome evaluation. The authors demonstrated that such involvement not only benefits the quality of care, but it also leads to increased departmental efficiency and therapist morale.

Still another legitimate role for respiratory therapists was suggested by Dr Albert Barrocas in his 1983 editorial, **A Role for Respiratory Therapists on the Nutritional Team**. Dr Barrocas contended that because malnutrition is such a significant factor in the development and treatment

of respiratory diseases, respiratory therapists should be members of nutritional support teams—and he challenged the therapists of the 80s to expand their roles in that direction. The gauntlet has been picked up in some settings, but not with the vigor that Dr Barrocas hoped for. A benefit of reading such a paper is that it draws our attention to options not fully explored and potentials not reached.

The March 1985 issue of RESPIRATORY CARE documented yet another role for the respiratory therapist. In the article, **A Role for the Respiratory Therapist in Flexible Fiberoptic Bronchoscopy**, Coppolo and colleagues described a study demonstrating the efficacy of utilizing therapists to provide bronchoscopic assistance to pulmonary internists. With this article, we see the role of the therapist expanding into diagnosis other than in the pulmonary function laboratory—and we see an even closer relationship with the physician.

The last of our ten republished papers specifically addresses the important role of the respiratory therapist in a highly specialized arena—neonatal respiratory care. Robert Perlson's article, **Safe and Successful Delivery-Room Intubation and Resuscitation of Meconium-Stained Newborns by Respiratory Therapists**, was published in August 1986. Mr Perlson described a study that analyzed the effectiveness of using respiratory therapists to identify infants with meconium-stained fluid, to determine the need for intubation and resuscitation, and to perform those procedures in the delivery room. The data supported the expansion of the therapist's traditional role in neonatal respiratory care to include the provision of these life-support procedures in the delivery room itself.

And Now to Your Reading

I hope you will enjoy reading these republished articles from the 1960-1986 period as much as I have. But let me remind you that these ten are only a sample of the myriad contributions to INHALATION THERAPY and RESPIRATORY CARE that trace our evolution as respiratory care practitioners over the past 35 years. I invite you to seek out back issues and read still more about our professional heritage. As professionals, we should not be content with knowing only the roles we play in health care today. We should take time to look back at our past struggles and successes, our ideas, good and bad. Without such reflection, we cannot fully appreciate the changes that have occurred over the years and the advances that we tend to take for granted today. And, just as important, without knowing the past we won't fully benefit from the lessons taught by those who preceded us—lessons that can help us negotiate our way as still more changes in our health-care role occur in the future.

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Therapists must become more expert as use of respiratory aids grows

by William C. Allen

THE GROWTH OF inhalation therapy as a paramedical specialty makes it obvious that the trained therapist will be an "expert" in respiratory aid techniques and will likely, in no small number of instances, be "the expert."

Already in some localities, inhalation therapists are *the* technicians with the knowledge and the ability to make application of the various mechanical respirators. There is apparently no question that the use of all types of mechanical respiratory aids is increasing and as this use spreads, along with the whole field of inhalation therapy, it well behooves the inhalation therapist to have a working knowledge of all such equipment.

Tank Respirators. Most everyone is familiar with the iron lung or tank respirator. The iron lung is generally considered to provide the most efficient form of artificial respiration so far as the mechanics of breathing are concerned. However, it is a large, bulky piece of equipment, hard to move, and leaves something to be desired in the convenience of patient care. The iron lung is an air-tight enclosure in which the patient is placed with his whole body – with the exception of the head – enclosed, and with a sealing collar around the neck. Pressures within the tank are varied by a diaphragm or bellows-type pump. When a sub-atmospheric pressure is created within the tank, this causes the thoracic cage to expand, accompanied by lung expansion which causes the patient to inhale. Usually the pressure in the tank is then reduced to atmospheric pressure and normal elastic forces cause exhalation. Positive pressures inside the tank can be provided to squeeze the thoracic cage and thus aid exhalation. Pressures used are adjustable through mechanical valving devices and rate of respiration is adjustable through variable speed drives to the pumping unit. Hand hole ports with sealing devices and transparent viewing ports are provided in the tank to make it possible for limited nursing care to take place while the tank is breathing the patient. Plastic domes are also provided for enclosing the patient's head in an air-tight enclosure while the patient is slid out of the tank for care. For this operation the

William C. Allen is a technical representative for Thompson Engineering Products, Inc., at the Boulder, Colo., office.

patient is breathed by positive pressure to the dome.

Self-contained 120-volt generating units or battery-operated inverter units providing 120 volts can be used to operate the tanks, making it possible to move them from one place to another without being attached to a wall outlet. Light-weight, battery-operated, portable pump units also can be used to operate the tanks in lieu of their regular pumping units to provide movability.

The iron lung—generally considered to provide the most efficient form of artificial respiration—does have its drawbacks. It is large, bulky, hard to move, and somewhat inconvenient for the best patient care.

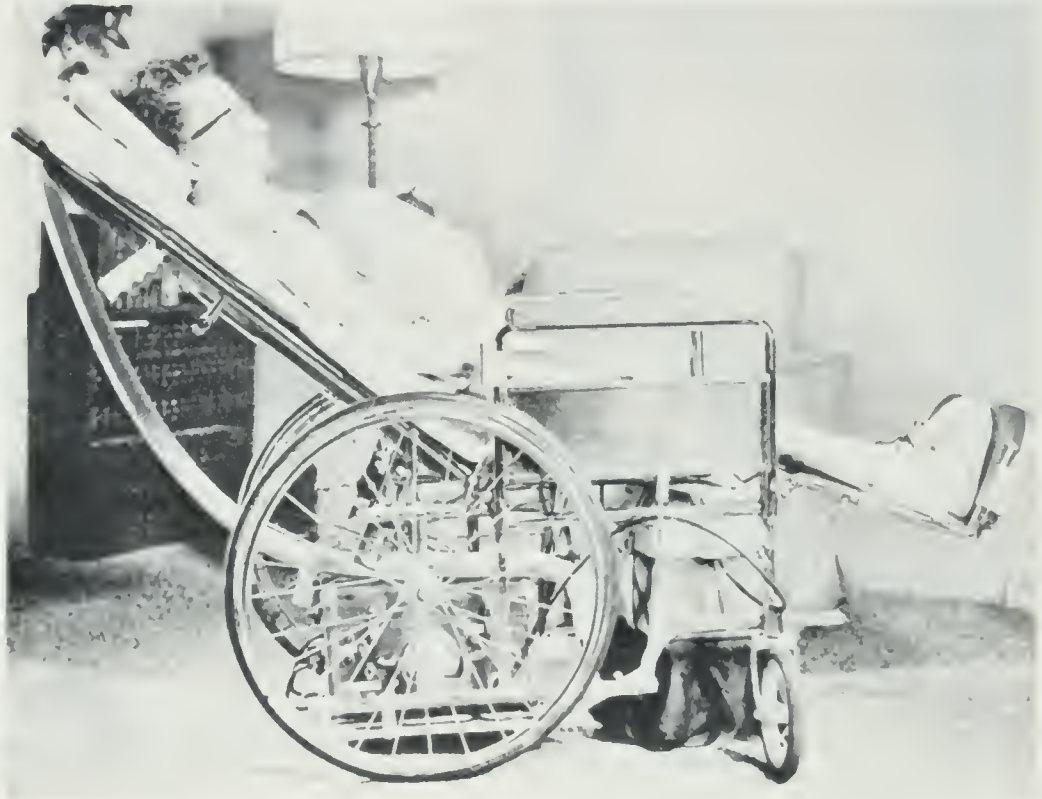
Light-weight half tanks with stretcher handles, using a separate portable pumping unit, also have been used to provide a fairly high degree of portability for a patient in a tank. These half tanks have become much more usable with the introduction of truly light-weight, dependable, portable pumping units which operate from automobile batteries.

Chest Type Respirators. A newer, smaller, and more convenient type of respirator is the chest shell respirator. This consists of a chest shell, or cuirass, which is strapped to the patient covering his chest or chest and part of his abdomen. There is a space between the shell and the body which is sealed around the outside with a sponge rubber or air-filled sealing device. A separate pumping unit attached to the shell through a flexible hose varies the pressures inside the shell with relation to atmospheric pressure in a rhythmic manner similar to the way pressures are varied in a tank. In the same way that a tank works when the pressure in the shell is reduced below atmospheric pressure, it causes expansion of the chest wall and thereby inspiration. Change then to atmospheric pressure or above atmospheric pressure inside the shell permits the thoracic cavity to contract or helps it to contract and cause expiration. Pressures used, as well as rate of respiration, are adjustable in the pumping unit. The cuirass respirator can be used by a patient who is in a regular hospital bed and also in most cases by a patient who is sitting up in a wheel chair or in an automobile. The advantages over the tank respirator in convenience are obvious.

When chest shells were first used the pumping units were fairly large and bulky and although some of them were called "portable" because they would operate from a battery power source, they still did not provide much greater ease for moving the patient about than did the tank. However, in the last three or four years, cuirass pumping units which are truly portable have been developed. These machines are light in weight and are small enough in size so that they can be mounted together with the battery for operation directly under a wheel chair (even a child's wheel chair). They can be easily carried and used in an automobile and they operate from either 120-volt house current or 12-volt battery. Chest shell, or cuirass, pumping units may be reciprocating units using the piston, or bellows-type pump, or rotary units using the air turbine, which is the type of pump you are accustomed to in a vacuum cleaner. The smallest and lightest weight units all use the turbine.

Battery Operation. For several years light-weight plastic battery cells have

been used to operate portable respirators because the cells are non-spillable, light in weight, and have small colored balls whose position indicates the state of charge. Lately, however, more and more regular automobile batteries are being used to operate these machines because they are much cheaper in price and because they give longer operating times. Then too, since most automobiles today have 12-volt electrical systems, it is proving most convenient to operate the respirators from 12-volt sources.



The cuirass respirator, which covers the chest and part of the abdomen, can be used by a patient who is in a regular hospital bed and, in most cases, by a patient sitting in a wheel chair or an automobile. Small, light-weight, truly portable pumping units have been developed to use with the chest respirator.

Some of these portable respirator pumps operate from 24-volt rather than 12-volt battery systems in order to provide longer battery operating times. They, of course, will not operate from a 12-volt automobile battery system, although they will operate from most airplane battery systems. The 12-volt battery units also operate from the 24- to 32-volt airplane battery systems when used in conjunction with a proper voltage regulator. A 12-volt machine on a long-life automobile battery will run just as long as any of the machines operating on 24-volt batteries.

Positive Pressure Respirators. The pumping units used for operation of the cuirass also all can be used with the proper attachments for positive pressure breathing. They provide a machine-controlled intermittent flow of room air, pressure and rate adjustable. They can be used for breathing a patient through the mouth by means of a mouthpiece or through the nose and/or mouth by use of a mask, or through a tracheostomy tube. *Efficient humidification of the air stream must be provided when breathing a patient through a tracheostomy because such*

breathing by-passes the normal humidification of the upper respiratory system.

Pumping units of a similar nature utilizing the air turbine but still smaller in size and lighter in weight have been developed for use in positive pressure breathing only. These units can be used for positive pressure through mouthpiece or face mask and also through tracheostomy tube. As in the cuirass-type respirators, these machines control the rate and depth of respiration, both of which are adjustable to the requirements of the individual patient. These machines are useful for breathing those patients who are unable to breathe by themselves or who find it difficult to ventilate satisfactorily by themselves but who are able to cooperate to the extent of relaxing so the machine can do the breathing for them.

There are patients who have sufficient breathing power of their own and are uncooperative to the extent that they fight this type of apparatus and for them it may be difficult or impossible to use. It is for such patients as this that the patient-cycled demand valve type of positive pressure breathing apparatus with which most inhalation therapists are already completely familiar is most useful. Several of the patient-demand type valves which operate from a high pressure compressed air or oxygen source also have automatic cycling devices incorporated. With these valves you can adjust both pressure and rate of respiration and breathe a patient with positive pressure in much the same manner as you would breathe him with one of the turbine type respirators.

A further development of the turbine-type positive pressure device is represented by those machines that provide a continuous flow of air with adjustable pressure and flow rate and which must be cycled either by the user or an attendant. Techniques of smoking like a pipe, and tongue or finger cycling, are used. The patient using this type device can completely control both the rate and depth of his respiration. Because of their simplicity these devices can be made much smaller and lighter in weight than any of the others previously described. They also are made to operate on either 120-volt house current or 12-volt battery. They are used principally as therapeutic devices for positive pressure therapy, deep breathing, or lung stretching exercises, and cough assistance. However, in some areas, this same type of apparatus is used for positive pressure breathing, patient controlled, over fairly long periods of time, i.e., for several hours.

A development stemming from this last described apparatus is a combination of the continuous air turbine with a second pump of the diaphragm-type making provision for the use of aerosolized medication along with the positive pressure. Through this device IPPB is provided, being made intermittent either by the patient or an attendant. Where intermittent positive pressure therapy using room air is desired this type of apparatus provides a highly convenient and portable as well as economical arrangement. Some of the patient-demand type valves also are provided with pumping units providing room air in order to make their operation more economical and in some cases somewhat more convenient.

Abdominal Belt Respirator. Probably the newest form of mechanical respiratory assistance is provided by the inflatable abdominal belt. Although the crude forerunner of this device was successfully used in England 25 years ago, it is only in the last two or three years that it has been used to any extent in this country. This device consists of a corset-like belt which is strapped around the abdomen of the patient extending a little over the lower ribs. Inside the front of this belt covering the abdomen is a balloon-like inflatable bladder. A separate pumping unit similar to those described above is used to alternately inflate the bladder and permit it to exhaust. When the bladder is inflated the abdominal contents are squeezed inward resulting in an elevation of the diaphragm causing exhalation to take place. When the air exhausts from

the bladder, the abdominal organs sag down allowing the diaphragm to drop, thus assisting inhalation. This device depends to a varying extent on the forces of gravity acting on the abdominal contents to pull the diaphragm back down, so that very few patients have received adequate ventilation from it in a prone position. Some pumps, of the piston type, provided for the abdominal belt respirator operate from both 120-volt house current and 24-volt battery and can be used for positive pressure breathing and for pumping the smaller sizes of chest shell as well as for

The abdominal belt respirator powered by portable batteries means greater mobility for the patient.

pumping the belt. Considerably smaller and lighter pumps, of the turbine type, operating from 120-volt house current and 12-volt battery are available for pumping the belt and also for positive pressure breathing.

Rocking Bed. The rocking bed, although dissimilar in operation from the apparatus described above, also is a mechanical respiratory device and should be included here. The rocking bed rocks back and forth like a see-saw and the rate of rocking, as well as the extent of the excursion from horizontal at the foot and at the head, is adjustable. When the feet of the patient are down, the abdominal contents pull the diaphragm down and cause inspiration. When the head is down and feet are up, the abdominal contents falling in the other direction push the diaphragm upwards in the thorax and cause expiration. Many people are quite satisfactorily ventilated as they sleep on the rocking bed.

Undoubtedly development of mechanical respirators and respiratory assisters will continue and the progressive inhalation therapists not only will want to become familiar with those now available, but keep abreast of future developments.

AAIT CHAPTERS

The American Association of Inhalation Therapists now has 21 chapters, the most in its history. Here they are with their headquarters city:

CHAPTER	CITY
Boy County (Michigan)	Bay City
Baston	Bostan
Cannecticut	New Hoven
Delaware Valley	Wilmington
Florida	Miami
Greater Montreal	Montreal
Greater New York	New York City
Illinois	Chicago
Michigan	Ann Arbar
North Texas	Dallas
Ohia	Cleveland
Rocky Mountain	Denver
Southeast Texas	Port Arthur
Southern Califarnia	Covina
Southern Ontario	Toronto
South Texas	Haustan
Tennessee	Nashville
Upper Midwest	Minneapolis
Western New York	Buffalo
Washington, D.C.	Washington
Wisconsin	Milwaukee

Additional chapters are being formed.

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Sister M. Teresa is a Certified Registered Nurse Anesthetist at St. Elisabeth Hospital, Granite City, Illinois. She is a graduate of St. Louis University College of Nursing and Barnes Hospital School of Anesthesia in St. Louis; a member of the American Association of Nurse Anesthetists, and a Life Fellow of the Seminars on Hypnosis Foundation. She is also an Associate member of the AAIT.

Consider Not Only The Therapy, But The Patient As Well!

by Sister M. Teresa

UNCONTROLLED fear in a patient already dyspneic from physical causes can evoke a vicious circle of paralyzing emotions, rendering an otherwise cooperative patient intellectually helpless, or even injurious to himself:

FEAR - BODILY EFFECTS - REALIZATION OF FEAR - ANXIETY - FURTHER CONSIDERATION OF BODILY EFFECTS - MORE TREMULOUSNESS - MORE FEAR, etc.

Since inhalation therapy is often a life-saving treatment, the emergency nature of the procedure is potentially frightening to the patient, because people tend to fear the unknown. We can tolerate unpleasantness if we expect it, know it is inevitable, and anticipate beneficial results – especially if we know how to control our reaction somewhat. But not knowing what to expect engenders tension, fearful anticipation, and an inability to cooperate.

Many patients in need of inhalation therapy are apprehensive to say the very least. Whether this apprehensiveness increases or disappears depends largely on the therapist's WORDS, ACTIONS AND MANNER. The patient will listen to every word the therapist says, and will place his own interpretation on it, which makes the choice of words by the therapist most important. The patient will watch the therapist's every move, and will readily discern either self-assurance or timidity. The patient needs to know that the therapist is qualified, that he is really interested in the patient and nothing else for the time; and he also needs to know just how much more discomfort is to be anticipated.

As a rational being, the patient has a right to have his treatment explained briefly in terms meaningful to him, thereby enlisting his intelligent cooperation. Terms meaningful to an ill, anxious, and therefore self-centered patient include answers to "What's this going to feel like?", "What will it do to me?", and "What am I supposed to do?"

For instance, when placing a dyspneic, perspiring patient in an oxygen tent, one might include in the explanation something like, "You'll feel refreshed in this tent. Your breathing will be eased, and we'll adjust the temperature to suit your comfort. You'll be able to relax in an abundance of fresh oxygen . . . I'll stay with you till you're more comfortable, and we'll check you often to be certain that everything's all right. We'll keep your doctor informed of your condition."

A distressed patient will be greatly relieved to hear REFRESHED, EASED, COMFORT, RELAX, FRESH, I'LL STAY WITH YOU, CERTAIN, ALL RIGHT, YOUR DOCTOR—particularly if these comforting words are spoken by a

therapist who obviously knows what to do and who handles the patient gently.

When referring to an oxygen catheter, one can call it a smooth little tube "to carry the oxygen right where it will be picked up by every breath you take." This is soothing in contrast to "that horrible hose in my nose" that the patient may have heard about previously.

A patient about to receive intermittent positive pressure breathing treatment will be happy to hear that he can relax and let the therapist and the machine help him to breathe deeply, easily. "You can relax and let us work for you. You know, relaxing requires no energy output; and you

Time taken to explain the operation of equipment is repaid by a reassured, cooperative patient and more effective therapy.

— Photo courtesy of Air-Shields, Inc.

can't force relaxation, but then, you don't have to. Just *let* yourself loosen up. Make like a wet noodle and you can enjoy really easy breathing. Lots of oxygen will come through here and go all the way into your lungs. You will feel them being filled with this fresh air, which comes back out again all by itself. You don't have to do anything but lie here and be lazy. All you smell is the nice clean plastic, or this soft cushion (mask)."

The words RELAX, ENJOY, CLEAN, SOFT are welcome words, conveying the idea to the patient that the therapist is interested in the patient's welfare. Concomitantly giving undivided attention to the patient's needs is an effective therapy synergistic with the oxygen, since it inspires confidence and tranquillity.

Consider too the wretched feeling the patient develops when two therapists enter the room and it becomes apparent that one is a beginner, who is going to secure "experience" at the patient's expense. Would it not be better for all concerned if the "beginner" were not so labelled in the patient's opinion, and if the patient were not made to feel himself to be a practice object, subject to a beginner's mistakes? The teaching could be done before seeing the patient, and any further explanations could be worded as if the *patient* were receiving instructions.

Also, what patient would not be righteously indignant if upset by two people "working on" him (or on some equipment near him), and talking simultaneously to each other about last night's late-late, or "My 'horrorscope' says I should be careful today or I might hurt somebody." Even scientific discussions are objectionable if they exclude THIS PARTICULAR PATIENT'S IMMEDIATE INTERESTS. An acutely ill patient does not care if what the therapist is doing for him will be of benefit to future patients by accumulating data to prove or refute some theory. If the same patient were well, he might cooperate in research projects, but to a sick person, these are "experiments" using him as a "guinea pig." Because the patient is ill, he wants and *needs* tender, loving, effective care RIGHT NOW.

A hypoxic individual, typified by a drowning person who will grab for any straw offering the least hope of rescue, is overwhelmed by the urgency of his oxygen needs. Let us say he is self-hypnotized by his own fears. He will therefore respond instantly and literally to suggestion, which can be verbal or non-verbal, but must have a *POSITIVE* aspect.

Recalling the vicious circle mentioned previously, the therapist must first take advantage of the patient's unusual aware-

ness of his own inner feelings (of fear) and change these feelings immediately to those of relaxation and confidence. This is done by well-chosen words, sincerely spoken, by one whose sole interest at the time is THIS PATIENT.

Whatever conveys to the patient the idea, "I'm going to help you now," will elicit relaxation. The usefulness of positive thinking is something to believe in and act upon, but not to talk about. If a patient erroneously concludes that I say he will begin feeling better *only* by his thinking so, then no matter how short of breath he is, he will have enough breath to tell me where to go!! But using the positive approach as a basis, one can help the patient accept therapy in a relaxed frame of mind, with dividends in increased comfort.

If, in utilizing sympathomimetic amines, a tremulousness or passing discomfort is to be anticipated, this should be mentioned. Likewise, the effective action of the Cofflator should be explained as "packed with power"—to soften the surprise when the cough comes.

However, words connoting pain, tension, difficulty, choking, gasping, and feeling badly are definitely always to be avoided. Simple phrases like LOTS OF FRESH AIR FOR YOU, SOON FEELING MORE COMFORTABLE, BREATHING E-A-S-I-L-Y, have a tremendous therapeutic effect when properly combined with application of technical skills and the physician's other prescribed therapies.

Restlessness, one of the manifestations of hypoxia, is relieved by oxygen; but it is relieved more quickly and effectively by oxygen plus kind words and gentle handling. This is true even if the patient is apparently unconscious. Anesthetized, sleeping, comatose, and semi-conscious patients may not consciously recall what they heard while apparently deaf to all sounds, *but they DO hear!* Though they may not be physically capable of reacting to it at the time, people hear *not* what is *meant,* but *exactly what is said.*

Alarm clocks are used universally on the premise that we hear when asleep—not saying we remember having heard, or that we respond to what we hear at the time. Another fact of which the therapist should be cognizant is that a drowsy and ill person is *humorless* and *unable to reason* clearly *or to act* energetically. Consequently, an "unconscious" patient, hearing attendants say, "He looks like Death warmed over," will not necessarily give any evidence of having heard; he will NOT take the remark as a joke, and will NOT know that it is being said about the passing interne who was up all night on maternity. This patient then begins to feel worse, and will show the effects symptomatically *later.*

It behooves the therapist to know the patient's general condition, as well as the pharmacology of all medications the patient is currently using, in order to understand various reactions, and to time his treatments to contribute to the patient's greatest comfort. Rapport and consultation with the medical and nursing personnel on the part of the inhalation therapist will result in optimum patient care, though it may mean sacrificing one's own time or prestige.

Speaking of time leads us to the discussion of the delightful challenge of therapy of children. The extra time spent in preparation will be amply repaid in smoother relationships and ultimately faster therapeutic results. Children detect the therapist's inner sentiments via the latter's WORDS, ACTIONS AND MANNER, even more readily than do adults. Little ones respond to game-playing and will accept the most grotesque equipment as *fun* if it is introduced properly: as an "outer space" gadget, or something his favorite comic character uses, or that the child will surely like.

Rules for successful therapy with children are: (1) Make friends first. (2) Play a short game, or just talk to discover his interests. (3) Do something simple and entertaining with him to help him into an accepting mood. *THEN* bring on the outer space helmet or Rube Goldberg contraption, and you and the other kid will have fun with it. Nine out of ten other kids anyway. . . .

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The Role of Patient Instruction in Improving IPPB Treatments

Ray Masferrer,* B.A.

INTERMITTENT positive pressure breathing (IPPB) is usually administered to patients in the belief that deep insufflation of the lungs obtains better distribution of nebulized medication.[1] In our hospital, where an average of 15,000 IPPB treatments are administered monthly, more than 75% of them are given with this objective.

The adequacy of ventilation in patients with normal lungs may be determined grossly by astute observation of the movement of the chest wall or more precisely by measurement of the tidal volume.[2] The Radford nomogram[3] indicates that the tidal volume of the average healthy young male is about 500 ml. It is our opinion that positive pressure for the purpose described above should provide tidal volumes of at least three to four times the Radford prediction, or 1500 to 2000 ml in average young males, for example.

In IPPB therapy it is usually important to maintain deep and slow breathing through the treatment. Cushing and Miller[4] have stated that when mist or medication is used, "slow deep breathing through a wide-open mouth yields maximum penetration of larger droplets into the depths of the airway." Because rapid breathing causes preferential distribution to the normal areas of the lung, deep slow breathing also facilitates gas and aerosol distribution throughout the lung, improving delivery to the obstructed areas. Usually such breathing is maintained only through continuous encouragement by the technician. Once the patient is left by himself, he frequently stops taking the treatment in the prescribed mannner. Thus, skill and understanding by the technician and cooperation by the patient are essential to make therapy effective. IPPB is not to be "given" to the patient; the patient should participate, understand the purpose of the treatment, and be taught to relax and breathe slowly and deeply. This must continue throughout the treatment. A correctly given treatment will usually be well tolerated.

According to Safar and Kunkel,[5] "While moderate hyperventilation is desirable in patients with healthy lungs, any degree of hyperventilation may become life-threatening in patients with chronic hypercarbia and hypoxia, as in emphysema with CO_2 narcosis." Hyperventilation during IPPB, with sharp lowering of CO_2 tension, decreases cerebral blood flow, causing dizziness, and also influences peripheral blood flow, causing numbness and tingling in the perioral region and in the fingers. Thus, it is apparent that the respiratory rate should be decreased to a minimum during IPPB to prevent these side effects.

When an inhalation therapy service is understaffed, technicians may be compelled to merely start each treatment, then leave the patient and return when the treatment time is over. Such a schedule does not allow time for sufficient instruction and continued coaching to insure that the patient uses the machine correctly; indeed, the patient may not use it at all after the technician leaves the room. The present study was conducted to evaluate the importance of patient instruction by the technician.

* Technical Director of Inhalation Therapy, Parkland Memorial Hospital, Dallas, Texas.

TABLE 1. *Tidal Volumes and Respiratory Frequencies during IPPB Therapy before and after One-time Instruction.*

Patient	Sex	Weight	Predicted Tidal volume (fr. Radford)	Measured tidal volume		Respiratory frequency	
				Before instruction	After instruction	Before instruction	After instruction
1	F	300 lbs	610 ml	200 ml	1500 ml	28/min	12/min
2	F	275	590	600	1100	20	10
3	F	133	380	500	1400	12	12
4	M	147	400	900	1000	32	12
5	M	157	440	600	1000	26	16
6	M	153	435	1200	1600	24	12
7	M	168	500	1000	1100	28	16
8	F	112	340	900	800	24	16
9	M	180	520	1200	1200	24	12
10	F	140	380	600	1050	20	16
11	M	160	460	700	1000	25	21
12	M	175	500	500	1000	20	12
13	M	210	580	1000	1700	24	14
Mean values		178 lbs	472 ml	762 ml	1188 ml	24/min	14/min

Method

Thirteen uninstructed patients were observed during IPPB treatments administered by mouthpiece. The respiratory frequency per minute (f) was recorded, and the tidal volume (V_T) was measured with a Wright respirometer during 10 expirations. The continuous medication nebulizer was shut off during spirometry so it would not falsely increase tidal volume readings. Then the treatment was interrupted and the patient was instructed in slow, relaxed abdominal-diaphragmatic breathing. Each patient was instructed to breathe only through the mouthpiece, to allow the respirator to inflate his lungs, to wait for the respirator to cut off with each inspiration, and to take sufficient time to exhale completely before starting another breath. Following instruction, the treatment was resumed, using the same respirator settings. Tidal volume and respiratory frequency were again measured.

Results

Table 1 lists the patients' sex, weight, predicted tidal volume according to the Radford nomogram, and measured tidal volumes and respiratory rates, both before and after one-time instruction. These data indicate that the goal of achieving three to four times the predicted tidal volumes during IPPB therapy was not accomplished, either before or after instruction. Before instruction, average tidal volumes were 61% greater than the norm for healthy adults. However, after instruction the average tidal volume rose to be 152% greater than normal. The respiratory frequency decreased an average of 41% after instruction. There was considerable variation from patient to patient. For example, patient No. 1 increased her tidal volume 650%, whereas patient No. 9 had no change in tidal volume but decreased her respiratory rate 50%.

Measurements of tidal volume and respiratory frequency were randomly repeated during the next two to five days, before and after reinstruction of the patients; see Table 2. It is apparent that some patients fail to continue adequate treatment after only one period of instruction.

Discussion

During this study we did not achieve the goal of increasing tidal volumes to three or four times normal. This was probably due to two factors. One, the patient population in our hospital consists largely

TABLE 2. *Tidal Volumes and Respiratory Frequencies during IPPB after Repeated Instructions.*

Patient	First Day Before-After Instructions		Second Day Before-After Instructions		Third Day Before-After Instructions		Fourth Day Before-After Instructions		Fifth Day Before-After Instructions	
	V_T*	f**	V_T	f	V_T	f	V_T	f	V_T	f
1	200–1500	28–12			1100–1500	17–12	1300–1400	14–12		
2	600–1100	20–10			750–1200	14–10	700–1300	16–10		
3	500–1400	12–12	1400–1400	14–11	1200–1400	14–12				
4	900–1000	32–12	800–1200	28–14	800–1150	26–18				
5	600–1000	26–16	700–1000	24–16						
6	1200–1600	24–12	1000–1500	12–12						
7	1000–1100	28–16			1100–1200	16–16			1300–	14–
8	900–800	24–16	850–900	24–14			900–1000	20–16		
9	1200–1200	24–12					1200–1300	20–11		
10	600–1050	20–16	700–1000	22–14	600–1000	18–16				
11	700–1000	25–21					900–1100	20–15		
12	500–1000	20–12	1000–1000	14–10	1100–1100	10–10			900–1250	14–12
13	1000–1700	24–14	1200–1700	20–14						

* V_T = tidal volume ** f = respiratory frequency (rate)

of seriously ill indigent persons with whom instructive communication is difficult. Better results could be expected in a private hospital. Two, the same respirator setting (20 cm H_2O pressure) was used for each patient regardless of the degree of pulmonary pathological derangement. Ordinarily, of course, respirator settings should be adjusted individually to achieve maximum benefits.

This study demonstrates that effective IPPB therapy can be achieved only with adequate patient instruction. Moreover, instruction and encouragement must be repeated during subsequent treatments, at least in some patients. Based on these observations, we feel that the efficacy of IPPB therapy should be evaluated by actual measurement of tidal volume and that the technician should continue instruction and respirator manipulation until the best possible results are obtained. Tidal volume measurements should be repeated during subsequent treatments to determine whether additional instruction is indicated. And, ideally, the technician should remain with the patient for the entirety of every treatment.

Summary

It was observed that deep slow breathing during IPPB treatments is maintained only through continuous encouragement by the technician and that better results are realized if he remains with the patient during the treatment. It was also found that repeated instructions are usually necessary for maintenance of adequate treatments. Tidal volume measurements during therapy provide useful evidence of the desired effects of instruction.

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Home Care for Pulmonary Diseases

M. Leone,* R.N., E. L. Cassara,** R.N., Myron Stein,† M.D.

THE number of patients afflicted with chronic lung disease in the United States has increased steadily during the past five years. In Rhode Island, chronic obstructive lung disease accounts for 31% of hospital admissions, compared to 16% five years ago.[1]

Medical and technical advances have made ventilatory care a complex problem which often leads to prolonged hospital stays. A recent patient was forced by the demands of the care regimen to remain in our Intensive Care Unit for nine months. The shortage of acute hospital beds occasionally necessitates early discharge, which in turn results in frequent readmissions. To cope with this problem and to attempt to break the cycle of admission-discharge-readmission, a home care program was established. The main objectives of this program are:

1. To shorten hospital stays for patients whose medical, nursing, and other needs can be met at home, and to discharge patients who otherwise would have had to remain in Intensive Care Units.
2. To reduce the demand for hospital beds by patients with chronic lung diseases.
3. To allow the patient to live in his own surroundings, where his emotional and psychological needs may be better met.
4. To allow the patient to rejoin his family as a functioning member.
5. To treat incipient respiratory failure and thereby prevent the need for hospital admission.

Prior to the establishment of the Home Care Program for Pulmonary Diseases, patients with chronic lung disease requiring permanent tracheostomies and the use of ventilators to control chronic respiratory failure were required to spend their days in Intensive Care Units. In these situations patients became passive as all their needs were met by the staff involved in their care.

During acute illness these surroundings were necessary, but with lesser degrees of illness these patients became depressed because of the restrictions and confinement. They were restricted by visiting hours and could not visit with younger members of their families. They were unable to maintain contact with the outside world via radio and TV and were surrounded by patients who were seriously ill. After several months in this environment, a patient might wonder if living were worthwhile.

Since the establishment of the Home Care Program, patients have been discharged to their homes where they can feel useful and enjoy more normal relationships. Ventilatory care, nursing services, and social and medical services are available to them through the program.

* Home Care Program for Pulmonary Diseases, Rhode Island Hospital, Providence, R.I.
** Technical Director, Ventilation Therapy Department, Rhode Island Hospital.
† Professor of Medicine, Brown University Medical School, Providence, R.I., and Medical Director of the Pulmonary Division, Rhode Island Hospital.

A patient can be discharged with a tracheostomy tube in place so a ventilator can be used when necessary. The patient and his family are taught to manage and change the tracheostomy tube, to dispense the nebulized medication, clean the equipment, and to recognize indications for the use of the ventilator.

On the day of discharge from the hospital, the nurse therapist visits the patient to assure him and his family that everything required for proper care is in the home. At the time of this visit any earlier in-hospital training of the patient and family members is reinforced. The patient's therapeutic routines are followed by the family under supervision of the Home Care nurse. The family is encouraged to call the Home Care Department at any hour of the day or night with questions that may arise. Following an initial home visit, daily support visits are scheduled by the nurse to give the prescribed chest physiotherapy and to reassess progress of the patient and the family situation.

Patients are instructed in the proper use and maintenance of equipment and in sterile techniques of airway suctioning to prevent infection.

Laboratory procedures including blood gases and pH measurements may be ordered by a physician on the same basis as he would order them in the hospital. Other members of the allied health care team may visit the patient as necessary.

To exemplify the effects of this program for a patient with many medical and therapy complications, let us review briefly the history of a 67-year-old retired baker with chronic obstructive lung disease who experienced progressive shortness of breath on exertion.

Case Reports

Case 1. The patient was admitted to the hospital eight times in a two-year span. On the ninth admission he had acute and chronic respiratory failure. Following unsuccessful therapy to improve blood gases, a tracheostomy was performed. Blood gases prior to tracheostomy were: Pa_{O_2}, 39 mm Hg; Pa_{CO_2}, 65 mm Hg; pH, 7.29; CO_2 content, 31 mEq/L. Following stabilization with prolonged ventilatory care, the patient was sent home with a tracheostomy tube in place. Again he did poorly, and four days later readmission became necessary because of increased respiratory distress and confusion. Blood pressure at that time was 100/60, respiratory rate, 30, and pulse, 120. His lungs revealed numerous rhonchi and wheezes throughout both fields. Sputum culture revealed a heavy growth of *Staphylococcus aureus*, coagulase positive. The patient was administered lincomycin medication. Prior to discharge he was referred to the Home Care Program. Since he has been on the Home Care Program he has performed well and has not required the ventilator most of the day, resuming some normal activities. He was readmitted approximately six months later for surgical revision of the tracheostomy. Blood gas studies showed that he still had chronic respiratory failure. Although he had severe right heart failure secondary to lung disease, he has been treated successfully at home. In the interim he has had three respiratory infections which probably would have resulted in hospitalization had there been no Home Care Program. Presently, a nurse therapist visits him daily for treatments and provides emotional support for him and his family. In good weather he is able to make brief excursions from his home, including a recent trip to a restaurant to celebrate his wedding anniversary. Were it not for the Home Care Program he certainly would have had a prolonged stay in the Intensive Care Unit of the hospital.

Some patients may require emergency admission to Home Care because deterioration of their respiratory status prevents continuing out-patient visits to a pulmonary clinic. Should such a patient appear to be in incipient respiratory distress, arrangements can be made to transfer him temporarily to the Home Care service. A brief history of a 70-year-old man with obstructive lung disease and chronic bronchitis with acute exacerbations will serve as an example.

Case 2. This man was followed at the out-patient clinic two times a week for surveillance, bronchodilator therapy, ultrasonic nebulization, and chest physical therapy. On two successive days he was unable to come to the clinic because of severe dyspnea accompanied by fever and increased coughing and sputum production. He was at this time placed on the Home Care Program for daily visits for five days. After the emergency treatment he improved sufficiently to return to his regular out-patient clinic regimen.

From experience with this type of problem prior to the establishment of our Home Care Program for Pulmonary Diseases, this patient with acute respiratory distress would have been admitted to the Accident Room as an emergency admission, had Home Care service not been available.

The criteria for admission to the program are:

1. Ability to meet medical, nursing, therapy, and emotional needs of the patient in the home.
2. Inability to meet these medical, nursing, therapy, and emotional needs of the patient in an out-patient facility.
3. Residence of the proposed patient within a ten-mile radius of Rhode Island Hospital.

Referral to the program is accomplished by written application by the private physician to the Home Care Committee. The decision for admission is the responsibility of the medical director who acts as Chairman of the Home Care Committee. Other members of the committee are private physicians, Home Care physician, hospital administrator, Blue Cross representative, Social Service representative, Ventilation Therapy Director, Home Care Director, District Nurse.

The referring physician remains responsible for overall medical care. Consultations from the Pulmonary Division are available to him at all times, but most particularly by way of the weekly conference of the Home Care Committee. Also at this weekly meeting each patient's progress is discussed. Individual plans for continuing therapy, discharge, or other disposition are made.

It has become obvious to the Home Care team that rehabilitation of patients with respiratory failure can be more successful at home than in the hospital. Some patients, unable to do much for themselves while hospitalized, are motivated by the home environment to attempt activities meaningful in their family life. These activities may range from smaller triumphs like shaving and airway suctioning to major accomplishments like stair climbing or a visit to the grandchildren.

The daily cost of the program per patient has steadily declined. Initially the cost was $43.00 per day to maintain a patient at home. While this figure contrasts sharply with the $100 a day to maintain the patient in the hospital, it does not compare to the current daily Home Care cost, which is only $9.00.

Blue Cross and Physicians Service, sensing the need for Home Care, have agreed to finance this program for one year. At this time 20% of our patients have Blue Cross coverage, 8% are Welfare recipients, and 72% are Medicare eligible.

When we review our results and compare them to our objectives we find that we have, indeed, reduced the demand for hospital beds by patients with chronic lung disease and have shortened hospital stays for patients who without Home Care would have had to remain in the hospital for indefinite periods. Most important of all, the patient has returned to his home surroundings where, free of rigid hospital routines, his emotional and psychological needs can be better met.

Perhaps the most important contribution of the Home Care Program is the status of the patients. By no means are they cured of their terrible and discomforting disease, but they are certainly happier at home.

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Training Respiratory Therapists in Emergency Endotracheal Intubation

Hewitt J Bryan ARIT
Chief Respiratory Therapist, The Roosevelt Hospital, New York City 10019.

Ronald A Andree MD
Associate Chairman, Department of Anesthesiology, and Medical Director, Respiratory Therapy Service, The Roosevelt Hospital.

EMERGENCY endotracheal intubation may now be considered one of the pertinent functions of the respiratory therapist. This report relates our experiences during the past three years in training respiratory therapists in this important skill.

We believe that therapists should be skilled at emergency intubation both because it is an important part of airway management and because trained physicians are not always available when emergencies occur. To amplify the first point, if therapists are to exhibit the high level of competence and confidence that we believe desirable, it is appropriate that they be able to handle any airway problem, from the initial life-threatening emergency to the final stages of ventilator adjustment. And the problem of the absence of physicians at emergency scenes is real even in our institution, a mid-Manhattan general hospital of 600 beds; anesthesiologists and nurse anesthetists are not always available, especially during the evening and night. Although our surgical residents may have learned the fundamentals of intubation during a four-week rotation in anesthesia, they too can be occupied with other responsibilities during these crises.

Our program of intubation instruction for respiratory therapists usually requires several weeks. The exact length depends on the opportunity to intubate patients in the operating rooms.

Trainees first attend a series of lectures by an anesthesiologist who reviews the topographic and internal anatomy of the airway. Particularly attention is directed to the landmarks of intubation, and models, films and slides are used to reinforce the lecture material.

After the lecture series, a nurse anesthetist or respiratory therapist discusses the use of masks, oropharyngeal airways, laryngoscopes, and endotracheal tubes. The trainees practice handling the instruments and perform intubations on plastic models of an infant and an adult.

Finally the trainees are given operating room experience. Supervised by an anesthesiologist or nurse anesthetist, each trainee is required to perform about ten successful endotracheal intubations in anesthetized, paralyzed patients. It is not unusual for the therapist to have some dif-

We are especially grateful to Elsie Svozil, CRNA, who directed the teaching in this program.

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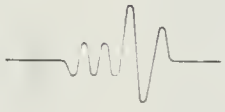
ficulty for the first two or three attempts and then to perform the next six or seven intubations successfully. In addition, the therapists look through bronchoscopes and esophagoscopes and also observe the heart and lungs during chest surgery; these experiences serve to fix anatomical relationships in their minds. Thereafter, therapists are permitted to institute emergency intubation. Because both therapists and house officers have been trained to intubate, the question of who should perform any particular emergency intubation sometimes arises. Our general policy is that the most experienced person should perform the task.

We kept records of the intubations attempted by our respiratory therapists during an 18-month period in 1969 and 1970. The airway emergencies ranged from cardiac arrests to accidental extubation of ventilator patients. In 79 emegencies, 71 patients were successfully intubated; an average of 1.3 intubation attempts was made per patient. Of the eight patients who were not intubated, two received successful emergency tracheotomy, one revived during the intubation attempt, and in five an airway could not be established because of excessive airway bleeding, secretions or vomitus.

Of the 71 intubations, 65 (91%) were performed by respiratory therapists, one by a nurse anesthetist, and five by surgical or anesthesiology residents after one or two unsuccessful attempts by a respiratory therapist. In two cases, reintubation was required because the tube had been placed in the esophagus.

Emergency endotracheal intubation skills can be taught to working clinical respiratory therapists. After training, they can, with a high degree of success, help to save lives in a variety of clinical emergencies.



The Effect of Home Respiratory Therapy on Hospital Readmission Rates of Patients with Chronic Obstructive Pulmonary Disease

Sue Roselle ACSW and Frank J D'Amico PhD

The South Hills Health System Home Health Agency, Homestead, Pennsylvania, studied the effect of home respiratory therapy on hospital readmission rates in 418 patients with chronic obstructive pulmonary disease (COPD). Respiratory therapists evaluated and followed referred patients in their homes. Oxygen, breathing equipment, and supplies were provided, and patients were educated in use, cleaning, and maintenance of equipment. Data for the study were taken from hospital records, home assessments, and discharge summaries. All patients studied had been hospitalized in the year prior to receiving home respiratory therapy. After 12 months of follow-up, 64% had not been rehospitalized. In the year prior to home respiratory therapy, the average number of hospital admissions per patient had been 1.28, with the average length of hospital stay being 18.25 days. During the home respiratory therapy study period of 12 months, the average number of hospital admissions was 0.48, with the average length of hospital stay being 6.09 days. These results indicate that home care provided by respiratory therapists can significantly reduce the rehospitalization of COPD patients. Prevention of rehospitalization in the study group resulted in estimated average hospital costs savings of $2,625 per patient for the year. As a result of this study, two large local industrial employers, the Federal Black Lung program, and Blue Cross of Western Pennsylvania have added respiratory therapy to their home health benefits. (Roselle S, D'Amico FJ. The Effect of Home Respiratory Therapy on Hospital Readmission Rates of Patients with Chronic Obstructive Pulmonary Disease. Respir Care 1982;27:1194-1199. Key words: Home care services, Hospitalization, Respiratory therapy.)

Ms Roselle is Executive Director, Emergency Medical Service Institute, Pittsburgh, Pennsylvania. When this study was performed Ms Roselle was Director of Allied Health Services, the South Hills Health System Home Health Agency, Homestead, Pennsylvania. Dr D'Amico is Assistant Professor, Department of Statistics, University of Pittsburgh, Pittsburgh, Pennsylvania.

The work was completed at the South Hills Health System Home Health Agency, Homestead, Pennsylvania. Research was funded by the Appalachian Regional Commission, Washington DC, with the technical assistance of the Southwestern Pennsylvania Economic Development District; the grant was administered by the United States Department of Health and Human Services. The pilot study of this project was funded by the Christmas Seal League of Southwestern Pennsylvania and by the American Association for Respiratory Therapy.

Reprints: Sue Roselle ACSW, Emergency Medical Service Institute, 3600 Forbes Avenue, Suite 206, Pittsburgh PA 15213.

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Introduction

As the cost of health care has risen over the past decade, emphasis has emerged on home health services as a humane and cost-effective alternative to acute care. This paper reports the cost-effective provision of home respiratory therapy to patients with chronic obstructive pulmonary disease (COPD) under the auspice of a certified home health agency.

Home respiratory care is not a new concept, but traditionally the care has been provided by a home health nurse as a follow-up to education conducted in the acute-care setting.[1] Malkus reported that hospital readmissions were prevented through the provision of nursing care to respiratory patients in their homes, but there was no statistical analysis of this finding.[2] Most research on this subject has focused on specific therapeutic modalities applied in the home, such as

oxygen[3] and intermittent positive pressure breathing.[4] Although various programs have been described,[5,6] the effect on rehospitalization rates of providing the services of qualified respiratory therapists in the home has not been reported in the literature.

The economic impact of COPD can be estimated in terms of the direct costs of treating patients and of reduced productivity caused by morbidity and mortality. A 1977 governmental estimate of the direct costs of hospital treatment, physicians' services, and prescribed drugs was $803 million. Costs in terms of lost earnings due to temporary or permanent disability were estimated to be $305 billion. Lost earnings due to mortality were estimated at $645 billion.[7]

These patients with COPD have been subjected to what has been described as "the dreary and depressing cycle of hospital admission/discharge/readmission."[8] Based on the belief that the services provided in the acute-care setting should be extended into the patient's home to break this cycle, the South Hills Health System Home Health Agency in Homestead, Pennsylvania, developed a respiratory service. A history of the project has been provided elsewhere by Cronin[9] and by Frownfelter.[10]

The South Hills Health System Home Health Agency offers a comprehensive and coordinated range of services in the patient's residence. These services include nursing, speech-language therapy, physical therapy, occupational therapy, social work, respiratory therapy, home health aides, and psychiatric nursing. Electrocardiograms, blood and other laboratory studies, pharmacy services, and health education are also provided. The agency arranges the delivery of all medical and surgical equipment and supplies to the home.

All the professional services provided by the home health agency are reimbursable by most health insurance companies, with the exception of respiratory therapy. Medicare will allow for the costs of using a respiratory therapist in an administrative or consulting capacity, but direct patient care by a respiratory therapist is not reimbursable on a per-visit basis.[11] To provide this essential service to patients in their homes, the South Hills Home Health Agency sought other sources of funding. The research described in this report was funded through a grant from the Appalachian Regional Commission, with the technical assistance of the Southwestern Pennsylvania Economic Development District. This grant, administered by the Department of Health and Human Services, expired September 30, 1981.

The primary objectives of the respiratory therapy service in this home health care project were identified as:

1. To provide respiratory therapy to those patients of the South Hills Health System Home Health Agency in need of this service.
2. To demonstrate that respiratory therapy provided in the home can reduce the number of hospital admissions and the length of stay when hospitalization is necessary for patients with COPD.
3. To provide statistical information to major health insurance carriers to permit them to recognize respiratory therapy as an essential home health service that reduces the cost of the treatment of respiratory disease.

Patients and Methods

Patient Population

Most patients are admitted to the home health agency following acute-care hospitalization. They are identified by members of the hospital staff or by their physicians as possibly being in need of home health services following their discharge. While the patient is still in the hospital, a nursing manager of the home health agency assesses the patient's eligibility for home health services.

The primary requirement for admission to the agency is the need for the services of a professional nurse, a physical therapist, or a speech-language pathologist. Additionally, the patient is required to have a physician, to identify a person to assume responsibility for his care in the absence of the agency staff, and to live within the geographic area served by the agency.

Once eligibility for home health services has been established, the individual professional services to be provided are identified either by the physician or by the agency staff in consultation with the physician. After the patient has been discharged from the hospital, a professional nurse makes an evaluation visit to the home, usually within 24 hours of hospital discharge. This evaluating nurse in turn refers the patient to other services in the agency.

Respiratory Therapy Services

The need for an evaluation by a respiratory therapist was determined either by the physician or by the home health nurse in consultation with the physician. Initially, many of the patients with COPD had been receiving other home health services and were then referred for respiratory therapy when this service became available. After the service was fully developed, most respiratory therapy referrals were initiated directly by patients' physicians.

Respiratory therapy in the home, like all services provided by a certified home agency, was required to be reasonable and necessary. Unless the patient's condition required the skilled intervention of a professional nurse, the patient did not qualify for the respiratory therapy service. The patient had to be visited by a professional nurse a minimum of once every 30 days to remain active with the respiratory therapy service.

The home health nurses and the respiratory therapists worked closely, coordinating visits and care plans to ensure that the patients received maximum benefit from the treatment regimens. The frequency and number of visits varied greatly, as determined by the needs of the patients and the respiratory therapy staff. Generally, the more ill the patient and the longer he had been known to the program, the greater were the frequency and number of visits.

The project staff consisted of three respiratory therapists who provided patient care and acted as consultants to other members of the home health agency staff regarding respiratory disease and treatment. All the therapists had had significant experience in the acute-care setting prior to their employment by the home health agency.

The major recognized methods of home respiratory therapy include patient education, bronchial hygiene, breathing retraining, physical reconditioning, oxygen therapy, postural drainage, and chest percussion.[8] Upon referral, a respiratory therapist visited the patient in his home to evaluate the medical history, respiratory status, use and care of equipment, and general understanding of the disease process. After the evaluation was completed and documented, goals were identified and a multidisciplinary care plan was developed. To assure that comprehensive home health care was available, the respiratory therapists were certified to perform arterial punctures to obtain samples for blood gas studies.

In addition to professional services, the patients were provided with oxygen, breathing equipment, and medical supplies. Use, cleaning, and maintenance of equipment was an important part of the patient education conducted by the respiratory therapists. The equipment prescribed for home use included standard aerosol nebulizers, ultrasonic nebulizers, humidifiers, respirators, and air compressors. The equipment and oxygen were ordered by the agency from medical equipment suppliers, who delivered the orders and provided the initial equipment use instructions. Cleaning procedures were standardized with area hospitals and with medical equipment suppliers to ensure that patients received consistent instruction from each person with whom they had contact.

Continued participation in the project was determined by the need of the patient and did not necessarily continue indefinitely. The reasons for a patient no longer to receive home respiratory therapy included:

- stabilization of respiratory condition
- understanding of the use of equipment and exercises, accompanied by appropriate self-care
- no longer being homebound
- no longer requiring respiratory care, as the condition was not chronic
- consistent refusal to comply with physician and respiratory therapist instructions

Study Methods

After a patient was referred to the home health service and evaluated by a respiratory therapist, data collection commenced. Initial information was taken from the hospital medical record, the home respiratory assessment, and the home health discharge summary. Additional information was collected about the patient's occupation, caretaker, smoking history, number of nursing and respiratory therapy visits, equipment, hospitalization history, hospital readmissions after entry into the home respiratory therapy project, source of payment, and primary and secondary diagnoses. The diagnoses were those identified by the patients' physicians at the time of admission to the home health agency and were subsequently coded by the current international ICD-9-CM method. In some instances, related diagnoses were grouped to simplify statistical analy-

sis. The date of the first respiratory therapy visit was used as the project admission date.

The home health agency's policy required that everyone identified as eligible to receive home respiratory therapy must have services equally available. It was therefore not possible to study the patients by randomly assigning them to two groups, one to receive home respiratory therapy and one to act as a control by receiving all services of the agency other than respiratory therapy. Therefore, we studied the numbers of hospital readmissions and lengths of stay experienced by patients after entry into the home respiratory therapy project in comparison to the numbers of hospitalizations and lengths of stay experienced prior to entry into the home care program. Hospital medical records for one year prior to each patient's project admission date were used as the source of the hospitalization experience for the 12-month period before the patient began to receive home respiratory therapy.

While the comparison of hospitalization experiences was to assess two 12-month periods, one prior to and the other during the home respiratory therapy project, the study included all patients referred to the service during the 25-month period between December 1, 1978 and December 31, 1980. Because some of the patients participated in the program for more than one year, while others were in the study only a few months, the reported statistics were calculated for various lengths of follow-up. The lengths of follow-up chosen were 3 months, 6 months, 9 months, and 12 months. If a patient was followed for 3 months only, this patient was included in the 3-month follow-up statistic only, while in the calculations of hospitalizations and lengths of stay prior to home respiratory therapy, the information for the total year was included.

Results

During the study period, 612 patients were referred to the respiratory therapy service for evaluation. Of these, 15 patients decided that further services were unnecessary following the first respiratory therapy visit. Another 44 patients were evaluated by the respiratory therapists as not requiring further care, primarily because these patients were well educated about their conditions and demonstrated high levels of expertise in the use of the breathing equipment in their homes.

The remaining patients were classified by the primary and secondary diagnoses identified by their physicians at the time of hospital discharge. Of the total cohort, 418 (68%) were classified as having a primary or secondary diagnosis of COPD. As this group comprised more than two thirds of the total population and no other diagnosis had so many representatives, this group's experience was then further examined. The results reported here pertain to this group of 418 patients with chronic obstructive pulmonary disease either as a primary or secondary diagnosis. There were two patients with lengths of stay prior to home respiratory therapy of 275 and 138 days who were eliminated from the statistical analysis, as these two extreme observations highly skewed the distribution. All calculations are based on the distribution without the information from these two patients.

The COPD patients were studied to determine what proportion of them were readmitted to a hospital during the study period, after they began to receive respiratory therapy in their homes. They were followed for 12 months, at the end of which 267 (64%) had not experienced rehospitalization, while 151 (36%) had been rehospitalized at least once.

The average ages of the two groups were 70.1 years for the non-readmitted group and 70.5 years for the readmitted group. The average number of hospital admissions during the year *prior* to the study period had been 1.25 times for the non-readmitted group and 1.32 times for the readmitted group. The corresponding lengths of hospital stay had been 18.36 days and 18.07 days, respectively. The statistics quoted showed no statistical difference between the readmitted COPD and non-readmitted COPD patient in the year prior to use of respiratory therapy. Since no prior differences were found, any differences after respiratory therapy could be attributed to respiratory therapy.

Table 1 compares the COPD patients' average numbers of hospitalizations and lengths of stay for 12-month periods prior to and *after* the start of home respiratory therapy. Figures are given at 3, 6, 9, and 12 months of follow-up. The 12-months follow-up figures show that the average number of hospital admissions during the home respiratory therapy study period was 0.48 admissions, compared to 1.28 admissions during the pre-study year. The average length of stay during the study period was 6.09 days,

Table 1. Average Number of Admissions and Length of Stay Prior to and Following Home Respiratory Therapy in Patients with Either Primary or Secondary Diagnosis of COPD.

	Prior to Respiratory Therapy	3 Months Follow-Up	6 Months Follow-Up	9 Months Follow-Up	12 Months Follow-Up
Average Number of Admissions	1.28	0.25	0.37	0.43	0.48
Average Length of Stay (days)	18.25	2.77	4.41	5.42	6.09

compared to 18.25 days in the year before the study. The figures at the longest period of follow-up (12 months) are significantly different from those prior to home respiratory therapy ($P < .01$) for both length of stay and number of admissions.

In addition, the figures for follow-up at 3, 6, 9, and 12 months appear to indicate a linear trend in both sets of data. The correlation for the number of admissions following home respiratory therapy is .96 and for length of stay is .98, both of these statistics being significant at $P < .025$. Although only four data points are being examined, leaving just two degrees of freedom to estimate experimental error, the percentages of variation explained by the follow-up periods are still high: 93% and 96%, respectively, for number of admissions and length of stay.

Discussion

This study examined the effect of home respiratory therapy on the number of hospital readmissions and length of hospital stay in 418 patients with COPD. The premise was that a patient receiving home care from respiratory therapists would be likely to experience reduced hospitalization compared to patients not receiving this service, with the ultimate result being a reduction in the cost of treating chronic respiratory disease. The South Hills Health System Home Health Agency was ideally suited to conduct this research because of the multiplicity of professional and paraprofessional services already available. All the care provided was interdisciplinary, with all necessary services available to all patients. Additionally, because of the agency's size, the study population was sufficiently large to assure that the results could be generalized.

The results of the study indicate that home care provided by respiratory therapists can significantly reduce the hospitalization of COPD patients. It is difficult to compare the cost of hospitalization prior to home respiratory therapy with the cost of home health services. However, in this study, after the cost of home care was taken into account, the prevention of rehospitalization resulted in estimated average savings in hospital costs of $2,625 per patient.

As a result of this research, respiratory therapy has been added to the home health benefits available to the employees and retirees of United States Steel, Consolidation Coal, the Black Lung Program of the Worker's Compensation Division of the United States Department of Labor, and Blue Cross of Western Pennsylvania. Home respiratory therapy was a recommended addition to Medicaid in Pennsylvania, but this program was unable to expand its benefits because of an anticipated change in Federal funding.

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Editor's note: The current administrator of the South Hills Health System Home Health Agency respiratory therapy program welcomes inquiries. Address Melissa Weimer RRT, Director of Respiratory Therapy, South Hills Health System Home Health Agency, 1800 West St, Homestead PA 15120.

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Review of a Bronchial Hygiene Evaluation Program

John R Walton MHA RRT, Barry A Shapiro MD,
and Calvin H Harrison BA RRT

In the 1970s the demand in our hospital for bronchial hygiene therapy (aerosols, IPPB, incentive spirometry, chest physical therapy) had increased to such a level that 20-30% of the ordered therapy was not being administered. Because the respiratory therapists and medical directors were convinced that much of the ordered therapy was unnecessary, the Respiratory Therapy Department began a program in 1978 in which specially trained respiratory therapists were authorized to evaluate all non-intensive-care patients for whom bronchial hygiene therapy had been ordered. The program protocol consists of a medical record review, a physical assessment of the patient, the development of a patient-care plan, and a re-evaluation every 2-3 days of the patient's continued need for therapy. We found that initiation of the program has led to improved documentation of the need for bronchial hygiene therapy and a significant decrease in *total* procedures performed, not merely a substitution of therapies. After being adjusted to the 1981 Consumer Price Index (CPI), total charges for bronchial hygiene therapy were markedly decreased even though hospital charges increased 77.4% *above* the CPI inflation rate. Since the program was begun, the respiratory therapy staff has been able to administer all ordered respiratory care services to patients in a critical care setting and not less than 90% of ordered bronchial hygiene therapy to patients outside the intensive care unit. House staff, attending physicians, and patients and their families appear to be satisfied with the therapist-evaluators, and the morale of respiratory therapists seems to have improved as a result of their being able to take a more active role in the treatment of their patients and to apply their skills to the patients most in need of them. (Walton JR, Shapiro BA, Harrison CH. Review of a Bronchial Hygiene Evaluation Program. Respir Care 1983;28:174-179. Key words: Aerosol therapy, Bronchial hygiene therapy, Incentive spirometry, Intermittent positive-pressure breathing, Physical therapy, chest, Respiratory therapy.)

Mr Walton is Administrative Director of Respiratory Therapy, Northwestern Memorial Hospital, Chicago, Illinois. Dr Shapiro is Professor of Clinical Anesthesia and Director, Division of Respiratory Critical Care, Department of Anesthesia, Northwestern University Medical School, Chicago. Mr Harrison is Assistant Director of Respiratory Therapy, Northwestern Memorial Hospital.

Reprints: John R Walton RRT, Respiratory Therapy Department, Northwestern Memorial Hospital, 250 East Superior, Chicago IL 60611

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The Respiratory Therapy Department of Northwestern Memorial Hospital (NMH), the main teaching hospital of Northwestern University Medical School, provides a full range of services in the 887-bed hospital and in the 170-bed Rehabilitation Institute of Chicago, which is affiliated with Northwestern University Medical Center. In the 1970s, though the department had increased its staff, the demand for services had also increased to such a level that by the mid-1970s a crisis had developed: There consistently were more orders for therapy than the staff

could perform. Also, at that time the benefits of some forms of respiratory therapy were called into question.[1,2]

Early in 1977, 20-30% of all bronchial hygiene therapy ordered (eg, aerosol, IPPB, incentive spirometry, and chest physical therapy) at NMH was routinely not being administered. The respiratory therapists and medical directors of the department were convinced that much of the ordered therapy was not necessary. Despite several attempts by the medical directors to influence the ordering patterns of other physicians, little documentable progress was made in decreasing unnecessary bronchial hygiene orders.

To address this problem, the department began a program in 1978 in which specially trained respiratory therapists were authorized to evaluate all non-intensive-care patients for whom bronchial hygiene therapy had been ordered. In their evaluations the therapists were to focus on determining the proper classification of non-critically-ill patients and the appropriate bronchial hygiene therapy for their needs.

We undertook our study to determine whether this bronchial hygiene evaluation program has influenced the number of orders for bronchial hygiene procedures and whether the program has thus allowed the more efficient use of respiratory therapy department staff.

The NMH Bronchial Hygiene Evaluation Program

Development of Program

Initially, a small group of therapists was trained to classify non-critically-ill patients by reviewing their medical records and performing physical examinations and then developing patient-care plans based on that classification and the patient's need for therapy. In the medical-record review, the therapist would look over data about the patient's recent temperature, his weight, pulse, blood pressure, respiratory rate, and activity level, the last 48 hours of progress notes, and the results of radiographs, pulmonary function tests, and any other pertinent laboratory tests. To perform the physical examination, the therapist would make a general observation of the patient, noting his breath sounds and whether he could cough and produce sputum. In developing a patient-care plan, the therapist would make his recommendations for treatment.

Description of Program

Within 8 hours of receipt of an initial order for bronchial hygiene therapy for a non-intensive-care patient, the therapist-evaluator attempts to assess the patient and make recommendations for treatment. To devise a treatment plan with appropriate therapy modalities and frequencies of therapy administration, the therapist-evaluator uses a patient-classification system (Appendix I), which distinguishes between patients requiring treatment for *therapeutic* reasons (to treat an already existing pulmonary abnormality) and those requiring treatment for *prophylactic* reasons (to prevent a pulmonary abnormality) plus accepted criteria for various therapies.[3]

After making his assessment and recommendations for treatment, the evaluator contacts the ordering physician. The treatment plan is usually adopted, and the written evaluation and the proposed treatment plan are inserted in the medical record. When order changes are needed, they are taken via telephone by the evaluator and countersigned by the physician within 24 hours. Every 48 to 72 hours each non-intensive-care patient on bronchial hygiene therapy is re-evaluated by the same process. Patients usually progress from Class I to Class III, with their therapy tapered accordingly in both modality and frequency. The physician may elect to write an order for this tapering according to the treatment plan documented in the record.

The medical directors periodically review completed bronchial hygiene evaluation forms. In addition, audits are performed semiannually to assess whether the bronchial hygiene evaluations are being completed in accordance with department protocol.

Study Methods

Using selected data from 1975 through 1981, we retrospectively analyzed the effects of the bronchial

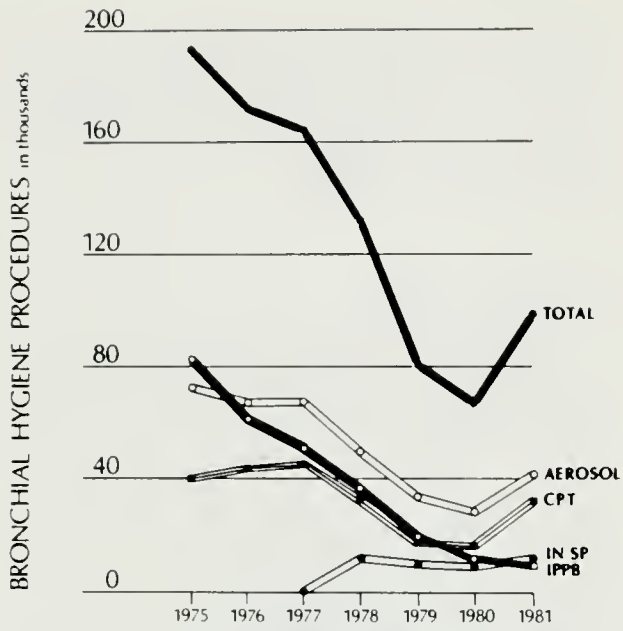

Fig. 1. Numbers of bronchial hygiene procedures performed at NMH from 1/75-12 81. AEROSOL = intermittent aerosol therapy; CPT = chest physiotherapy; IN SP = incentive spirometry; IPPB = intermittent positive-pressure breathing treatments. TOTAL = all AEROSOL, CPT, IN SP, and IPPB treatments. The bronchial hygiene evaluation program became effective in 1978.

hygiene evaluation program. (Data previous to 1975 were not included because there had been a reorganization of hospital services shortly before that time.) Data included demographic variables of the hospital, numbers of procedures performed, and revenue and expenses generated before and after the program began. Charges for bronchial hygiene therapy from 1975 through 1981 were adjusted to the 1981 Consumer Price Index (CPI).

Results

Total bronchial hygiene procedures decreased 49.1% between 1975 and 1981, with the most marked declines coinciding with the initiation of the bronchial hygiene evaluation program in 1978 (Fig. 1). Aerosol, IPPB, and chest physical therapy procedures decreased significantly after the initiation of the bronchial hygiene program (Fig. 1; Table 1). The only modality to increase was incentive spirometry.

Total CPI-adjusted charges decreased 9.7% between 1975 and 1981, with the decline occurring only after initiation of the bronchial hygiene evaluation program in 1978 (Fig. 2). These CPI-adjusted charges for bronchial hygiene therapy per patient day markedly decreased in spite of the fact that charges per procedure increased 77.4% *above* the CPI inflation rate. Correcting the total adjusted bronchial hygiene charges for this increase of hospital rates above the CPI yields an average decrease of $1,050,312 in charges per year after this program was initiated. Thus, if hospital charges had not increased above the CPI during this 7-year period, total bronchial hygiene charges would have decreased by approximately 46.3%.

Although patient charges processed by the department do not necessarily reflect the actual cost of providing services, charges are a useful indicator of the costs imposed on patients or third-party payers for provision of hospital respiratory therapy services.

As can be seen in Table 2, there were statistically significant changes in patient days, number of admissions, and the average length of stay at NMH

Table 1 Mean Numbers of Procedures Performed per Month at NMH during Periods Before and After Bronchial Hygiene Evaluation Program Was Initiated

Bronchial Hygiene Procedure	Mean Number of Procedures Month (± Standard Deviation)	
	Before*	After†
IPPB	5,407.80 (± 1,223.60)	1,625.00 (± 965.63)
Aerosol	5,748.00 (± 503.53)	3,221.70 (± 835.54)
Chest Physical Therapy	3,563.60 (± 498 60)	2,141.40 (± 796.07)
Incentive Spirometry	11.30 (± 30.08)	915.10 (± 374.25)
Total of All Procedures	14,730.70 (± 1,668.12)	7,903.20 (± 2,349.23)

$P < 0.0001$ for each "Before" and "After" procedure pair.

* "Before" refers to 36-month period (1/75-12/77) before evaluation program was initiated.

† "After" refers to 48-month period (1/78-12/81) after evaluation program was initiated.

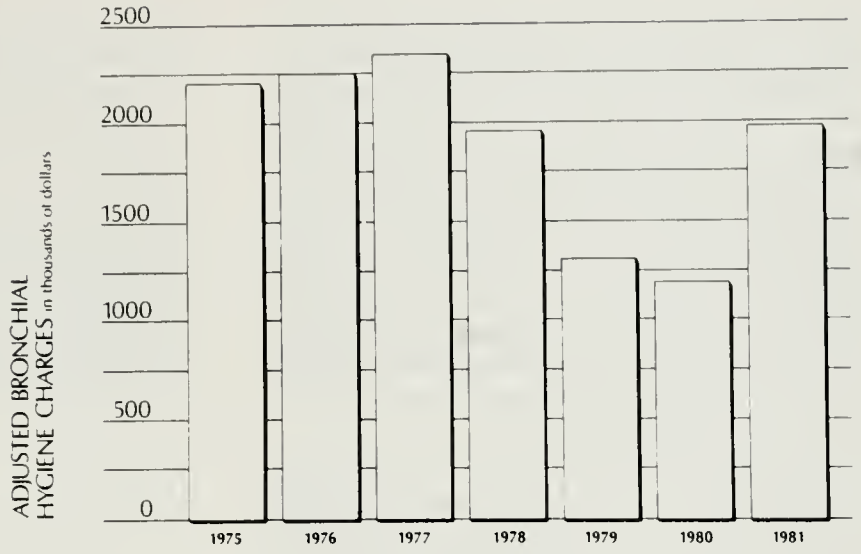

Fig. 2. Total yearly bronchial hygiene therapy charges, adjusted to constant 1981 dollars according to the 1981 Consumer Price Index. The bronchial hygiene evaluation program became effective in 1978.

after the evaluation program was instituted. However, the decrease in number of patient days (the product of admissions and length of stay) could account for only 4.6% (3,730 procedures) of the average drop of 81,390 bronchial hygiene procedures per year since the evaluation program began.

Based on 1981 data, the average time spent performing a bronchial hygiene evaluation was 21 minutes and the average time spent in making a re-evaluation was 18 minutes. New evaluators took noticeably more time performing evaluations and re-evaluations than did experienced evaluators. During 1978, 2,992 evaluations were performed, compared to 1,836 in 1981. In 1978, 3,368 re-evaluations were performed, compared to 1,955 in 1981.

After initiation of the evaluation program, the respiratory therapy staff has been able to administer all ordered respiratory care services to patients in critical care settings and not less than 90% of ordered bronchial hygiene therapy to patients outside the intensive care unit (ICU). As the orders for bronchial hygiene therapy have decreased, orders for acute care services have increased; thus, though rearrangements in staff assignments have been necessary, changes in budget have not. Currently, 60% of all respiratory therapists in the department, compared to 37% before the evaluation program began, work exclusively in critical care areas.

Table 2. Mean Numbers of Patient Days and Admissions per Month and Mean Lengths of Stay per Month at NMH during Periods Before and After Bronchial Hygiene Evaluation Program Was Initiated

	Mean Numbers/Month (± Standard Deviation)		
Period	Patient Days	Admissions	Lengths of Stay (Days)
36-Month Period Before*	23,725.80 (±895.56)	2,339.20 (±139.73)	10.170 (±0.414)
48-Month Period After†	23,225.20 (±851.42)	2,421.80 (±131.77)	9.600 (±0.334)

$P < 0.01$ for each "Before" and "After" demographic pair

* "Before" refers to 36-month period (1/75-12/77) before evaluation program was initiated.

† "After" refers to 48-month period (1/78-12/81) after evaluation program was initiated.

Discussion

Few complaints regarding bronchial hygiene therapy services have been received from physicians, nurses, or patients and their families since 1978. In

particular, both house staff and attending physicians appear to be satisfied with the therapist-evaluators.[4]

Although it is difficult to measure, the morale of therapists in the department seems to have improved since initiation of the program. We believe that this is because therapists now take a more active role in the treatment of their patients and are able to focus their attention on the patients who truly need their services. If our plan to train additional therapists as evaluators is accomplished, we will not have to dedicate a therapist to making evaluations on a particular day and evaluation can then become a normal part of a therapist's daily duties.

An expected advantage of the bronchial hygiene evaluation program was that respiratory therapy personnel would be better used, and we believe that respiratory therapy staff members are now applying their skills in areas where they are most needed.

Another expected benefit of the program was that the indications for and the effectiveness of bronchial hygiene therapy would be documented. Because of the completion of bronchial hygiene evaluation forms for each patient receiving bronchial hygiene therapy, both at the outset of therapy and at every 48-72 hour interval thereafter, indications for therapy are clearly documented and patient progress is noted.

The total cost to the hospital of providing the program is predominantly in the evaluation team's salaries and benefits. Initially, the large number of patients in the program required that two therapists a day be dedicated to performing evaluations (one therapist for each day and evening shift), 7 days a week. Because of the dramatic drop in bronchial hygiene therapy procedures ordered, more than one therapist a day dedicated to performing evaluations is seldom required. Based on 1981 data, NMH spends approximately $25,000 per year for a full-time therapist's wages and benefits; thus, the current cost of the program is approximately $40,000 per year (1.6 full-time-equivalent therapists).

Despite the substantial decrease in bronchial hygiene therapy in non-ICU patients, we have been unable to uncover any evidence that patient care has been adversely affected.

Some who have observed the decline in certain bronchial hygiene therapy procedures during a similar period have reported that a simple substitution of therapies has occurred.[5,6] However, we found that the *total* number of bronchial hygiene procedures declined after initiation of the bronchial hygiene evaluation program at NMH.

As can be seen in Figure 1, the total number of bronchial hygiene procedures increased in 1981. This increase slightly alters the magnitude of the previously reported effects of the bronchial hygiene evaluation program,[7] and we have begun a review of our program to discover whether other hospital demographic factors may have been responsible. At this time we have not determined why the number of procedures increased in 1981.

Summary

We conclude that the decrease in the number of patient days, the result of a decrease in admissions and length of stay, was not a significant factor in the decrease in the number of bronchial hygiene therapy treatments performed. To the best of our knowledge the only variable that could have influenced the number of treatments to this degree was the development of a bronchial hygiene evaluation program that relies on the skills of respiratory therapists. We believe that the development of similar programs in other hospitals can lead to improved documentation of the need for bronchial hygiene therapy, to a decrease in total numbers of bronchial hygiene therapy procedures ordered, to the more efficient use of respiratory therapy staff, and possibly to an improvement in respiratory therapy staff morale.

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APPENDIX I

Patient-Classification System

Class I

A. Rationale for Therapy

1. Patient has acute therapeutic indications, such as active pulmonary abnormalities that are treatable by bronchial hygiene therapy—in particular, retained secretions, recent segmental or lobar atelectasis of noncompression origin, and infiltrative disease with copious secretions.

2. Patient has acute prophylactic indications, such as having recently (within 48 hours) undergone one or more of the following:

   a. thoracic or abdominal surgery
   b. craniotomy
   c. laminectomy
   d. hip surgery
   e. radical mastectomy
   f. radical neck surgery

3. Patient has general indications such as the following:

   a. Recently (within 24 hours) transferred from ICU
   b. Hospitalized primarily for bronchial hygiene therapy
   c. Had received bronchial hygiene therapy as an outpatient or at home
   d. Has been identified by medical directors, fellows, residents, and medical students on the respiratory care physician service as one requiring close attention
   e. Has incentive spirometry orders only. (Applies to some patients only.)

B. Therapy Modalities and Frequencies

1. For a patient with therapeutic or general indications: Appropriate modalities are aerosol therapy, IPPB therapy, and/or chest physical therapy. Therapy frequencies usually required are three times a day (TID), four times a day (QID), or QID plus one treatment at night. Only under unusual circumstances is therapy as often as every 4 hours necessary.

2. For a patient with prophylactic indications: Typically, incentive spirometry only is required. After an initial instruction session is performed by a therapist, only one follow-up session is usually necessary. An exception to this is in the treatment of the acute high-lesion spinal-cord patient, who should receive incentive spirometry with therapist assistance at frequencies up to three times a day.

Class II

A. Rationale for Therapy

1. Patient has a need for therapy that is reasonable but less clear than that of Class I patients.

2. Patient was previously in Class I and therapy is being tapered.

B. Therapy Modalities and Frequencies

1. A stabilized high-lesion spinal-cord patient receiving only incentive spirometry may require the assistance of a therapist as frequently as twice a day.

2. Any other patients may require any of the bronchial hygiene modalities but at a frequency of TID or less.

Class III

A. Rationale for Therapy

Patient requires therapy once or twice a day and usually is on self-motivated incentive spirometry only.

B. Therapy Modalities and Frequencies

For the majority of these patients, incentive spirometry only is required. However, other bronchial hygiene modalities may be necessary but usually no more frequently than BID.

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A Role for Respiratory Therapists on the Nutritional Team

During the past decade, there has been increased awareness of the need for nutritional assessment and for treatment of malnutrition in hospitalized patients, including those with pulmonary disease and those who are ventilator-dependent. Surveys have indicated that 40-50% of the patients in U.S. hospitals exhibit some form of malnutrition.[1] The early recognition of these nutrition-related derangements has led to shorter hospitalizations, decreased incidence of complications, and an earlier return of patients to gainful employment.[2] The economic benefits of this decreased use of hospital resources are obvious.

In the past, malnutrition as a factor in the development and treatment of respiratory diseases had not been well-established or communicated. It has long been held that the most common cause of death in starvation is pneumonia; however, only recently have the various mechanisms that cause pneumonia during starvation been studied and applied.[3] Impaired macrophagic activity and decreased cell-mediated immunity play a role in the development of pneumonia, atelectasis, sepsis, and death in critically ill, starving patients.[4] Animal and human studies of malnutrition have demonstrated the development of emphysema, as well as hemorrhage and decreased levels of surfactant, both of which lead to alveolar collapse and atelectasis.[5] The loss of plasma albumin results in the lowering of oncotic pressure, which causes exudation of fluid into the interstitial and alveolar spaces. Decreased levels of serum proteins can also precipitate acid-base abnormalities.

Clinical studies have revealed that the nutritional support of patients on ventilators is often neglected, leading in a short time to malnutrition.[6] Malnutrition may be a critical factor in the difficulty of weaning patients from mechanical ventilation. The decrease in muscle mass during starvation causes decreases in tidal volume and vital capacity and poor cough reflexes, which worsen weaning difficulties. Nutritional support has been shown to increase the success rate of weaning ventilator-dependent patients.[7]

Metabolic complications experienced during enteral and parenteral nutrition can affect ventilator-dependent patients. Dehydration, volume overload, glycosuria, and hyperosmolar coma are well-known complications.[8] Phosphorus depletion, seen during nutritional therapy devoid of this element, can result in respiratory distress.[9] Recently, Askanazi et al[10] have shed light on a metabolic complication of interest to the respiratory therapist—the glucose overload syndrome. These investigators have found that patients receiving the majority of their calories as glucose tend to have an increase in minute volume ventilation, an increase at times approaching 100%.

Nutritional support does have potential pulmonary complications. Mechanical complications observed with the central mode of parenteral nutrition are associated with the catheter's introduction into the superior vena cava. Pneumo-, hemo-, hydro-, and chylothorax have been reported.[11] In patients receiving feedings orally or through a feeding tube, the potential for respiratory complications is inherent. Patients with endotracheal tubes have impaired swallowing mechanisms, which can result in aspiration. Patients with frequent cough can dislodge tubes into the esophagus, leading to aspiration.

Because they are so often involved in the care of patients on long-term mechanical ventilation and in the rehabilitation of patients with chronic pulmonary disease, respiratory therapists need to understand the interrelationship of malnutrition and lung disease. The therapist should be well versed in the various modalities of nutritional support and in their potential complications in relation to the cardiopulmonary system and thorax.

In the last 10 years, a health care team has evolved to aid in the assessment of nutritional health and the treatment of malnutrition in hospitalized patients. The multidisciplinary team usually includes physicians, nurses, pharmacists, and dietitians. I propose that the respiratory therapist can be an important member of the nutritional support team for the following reasons:

- Respiratory therapists frequently perform inspired and expired gas analyses and pulmonary function tests (eg, vital capacity, tidal volume, and compliance), which provide valuable information about a patient's state of nutrition and degree of stress. These measurements are also important in determining the amount and type of calories necessary for nutritional support.
- In close contact with severely stressed and critically ill patients, the respiratory therapist is in a position to make an early identification of patients who might be at risk for nutritional depletion.
- Because they often evaluate chest radiographs, respiratory therapists can help detect malpositioned parenteral lines and pulmonary complications of nutritional therapy, such as new infiltrates and hemo-, pneumo-, and hydrothorax.
- Computers are used to perform nutritional assessment and formula calculations and to record statistics. The availability of computer technology in a growing number of respiratory therapy departments gives this section of the hospital the potential to be the focal point for activities associated with nutritional support teams. The increased use of expensive computer technology for nutritional analysis makes the equipment more cost effective. It is feasible that the respiratory therapy department could develop into the cost-control center for the nutritional team.

During the past 5 years, the nutritional support team in our community hospital has benefited greatly from the participation of the respiratory therapy department. Initially the department's only contribution was computer support for the various determinations of nutritional assessment. Later, the department provided monthly statistics and participated in established educational programs. A quality-assurance study of nutritional assessments aided in improving the return time of these reports. The respiratory therapy department coordinated the activities of the nutritional team until a full-time clinical coordinator from the pharmacy department was appointed. The monthly nutrition newsletter is published by the respiratory therapy department. Plans for future activities include supplementing the computerized nutritional assessment with nutritional support recommendations. The possibility of O_2/CO_2-base indirect calorimetry is also being considered.

In our experience the presence of a respiratory therapist on our team has benefited all of us: The therapist has widened his horizon by encompassing nutrition in his medical knowledge, while the other members of the team have learned the importance of the respiratory system and the value of the respiratory department in nutritional assessment and support.

Nutritional therapy can be one of the challenges for respiratory therapists in the 1980s. To help therapists meet this challenge, educators must incorporate information on nutrition and nutritional therapy into respiratory therapy curricula. Continuing education should also be provided as information increases and techniques improve.

Albert Barrocas MD
Medical Director, Nutritional Support Service
Pendleton Memorial Methodist Hospital
Clinical Associate Professor of
Surgery and Nutrition
Tulane University School of Medicine
New Orleans, Louisiana
Medical Advisor
Tri-State Respiratory Therapy Conference

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A Role for the Respiratory Therapist in Flexible Fiberoptic Bronchoscopy

Dominic P Coppolo RRT, L Tarry Brienza RRT, David S Pratt MD, and John J May MD

All bronchoscopies in our institution are performed by physician-respiratory therapist teams. The therapist instructs the patient, anesthetizes him, does the biopsies, and prepares specimens for laboratory testing. Methods: We undertook a retrospective study of our performance to evaluate the safety, yield, and cost of this system. Results: Of 216 procedures evaluated, therapist-performed biopsies provided a specific diagnosis in 55%. When a tumor-like mass was seen directly, biopsies were positive 95.7% of the time. No procedure was associated with death. Complications included fever in about 9%, hoarseness in 2%, hemoptysis in 1%, and pneumothorax in one procedure (0.5%). Our institutional costs for flexible fiberoptic bronchoscopy by a physician-respiratory therapist team were only 34% of what they would have been if the same procedures had been performed in the operating room. Results performed were comparable to all others reported in the literature. Conclusion: This study clearly demonstrates that respiratory therapists can provide high quality, low cost, safe bronchoscopic assistance to pulmonary internists. (Respir Care 1985;30:323-327.)

Introduction

Since its introduction into standard medical practice in 1970,[1] the use of flexible fiberoptic bronchoscopy (FFB) has enjoyed an explosive growth. IMS America Ltd (a medical marketing research firm) estimated that 200,000 endoscopic examinations were performed in 1982. With billings to patients in excess of $400 per procedure, that amounts to more than $80 million in charges that year. The escalating rate of medical costs prompted us to develop endoscopy services outside the operating room, which carries the high price of a specialized area and staff. By using a trained respiratory therapist to assist a physician, we are able to avoid operating-room costs. The use of respiratory therapists, who can generate other revenues through respiratory therapy activities when not assisting at bronchoscopy, is far more efficient in our institution than it would be to hire at a lower salary scale a less qualified assistant, whose time would be entirely devoted to bronchoscopy.

The role of the respiratory therapist on this team is to educate the patient about the procedure; to administer the topical anesthetic prior to bronchoscopy; to collect all specimens; to perform biopsies; to monitor pulse, respiration, the ear oximeter, and the electrocardiogram when indicated; and to be responsible for the cleaning and quality assurance of the bronchoscope.

Mr Coppolo is Clinical Coordinator and Mr Brienza is Manager, Respiratory Care Services, Mary Imogene Bassett Hospital, Cooperstown, New York. Drs Pratt and May are Attending Physicians, Pulmonary Medicine, Mary Imogene Bassett Hospital, and Assistant Professors of Clinical Medicine, College of Physicians and Surgeons, Columbia University, New York City.
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Reprints: Dominic P Coppolo RRT, Respiratory Care Services, Mary Imogene Bassett Hospital, Atwell Road, Cooperstown NY 13326.

In order to assess the efficiency, safety, and economy of this system, we undertook a retrospective study of the records of all persons who had undergone bronchoscopy at our institution, where the procedure is performed by the physician-therapist team. We compared our data on complications and rate of positive biopsies to the data of other studies and contrasted our institutional cost for bronchoscopy by the physician-therapist team, outside the operating room, to what our institutional cost would be if the procedure were performed in the operating room.

Materials and Methods

For the purposes of this study, we reviewed the charts of all patients who had undergone FFB between January 1978 and June 1982. We used a standard format to gather information on the patient's age, sex, smoking history, clinical presentation, and chest radiograph findings. Also documented were complications occurring within 48 hours after the examination. The possible complications included fever, hoarseness, hemoptysis (greater than 100 ml), pneumothorax, bronchospasm, laryngospasm, myocardial infarction, and death. We compared our data to those of other studies that had looked at the same kinds of information. When needed, tests of statistical significance were applied. To compare proportions, we used the chi square test, with $P < 0.05$ indicating statistically significant difference.

In our system, the respiratory therapist first provides instruction to the patient regarding the administration of local anesthetic, likely sensations during the examination, and precautions to observe after completion of the procedure. (The anesthetic we use most often is 10 ml of 4% lidocaine, delivered by ultrasonic nebulization.[2] We use the ultrasonic nebulizer because we have found that it results in adequate anesthetic effect and is well tolerated by nearly every patient. Rarely, a patient is too sedated or is otherwise unable to cooperate in taking the ultrasonic aerosol, and for such instances we use a gas-powered atomizer. It has been our experience that both devices provide equally good anesthesia of the upper airway.)

When a biopsy of a directly visible lesion is to be done, the physician selects the appropriate site and maintains the distal tip of the bronchoscope in the proper position relative to that site. The therapist then passes the forceps to the end of the bronchoscope and, with the direction of the physician, advances the opened forceps and completes the biopsy. With a transbronchial biopsy performed through the wall of the bronchus or on a lesion not directly visible, the physician wedges the bronchoscope into the appropriate bronchus and maintains it there while, with fluoroscopic guidance and the physician's direction, the therapist passes the forceps and performs the biopsy. This avoids the physician's changing hand position and possibly displacing the bronchoscope. We find that this technique is much less cumbersome than is a one-person operation and that it allows the procedure to move quickly.

Patients excluded from this study were those who had undergone other invasive diagnostic procedures, such as transthoracic needle biopsy or open-lung biopsy, within 48 hours of the FFB examination, and those whose records did not contain sufficiently detailed data to permit an accurate retrospective evaluation.

Of 230 bronchoscopies performed between January 1978 and June 1982 by the physician-therapist team, the charts of 216 procedures, involving 201 patients, were evaluable by our study criteria.

Results

Our study population consisted of 139 men and 62 women. Fourteen patients were excluded from the total group of 230 who had undergone bronchoscopies during the 4½-year period. Because nine had had transthoracic needle biopsies and three had had both needle biopsies and open lung biopsies within 48 hours of the bronchoscopy procedure, we could not include them, as the other procedures could have produced complications similar to those of bronchoscopy. The other two patients were excluded because the record did not contain sufficiently detailed data to permit an accurate retrospective evaluation.

The mean age for men was 64, and for women, 59. The mean pack-years of cigarettes smoked was 51 for men, and for women, 32; 81% of the patients were smokers. Cough, increased sputum production, weight loss, and hemoptysis were the most common presenting signs and symptoms (Table 1).

A biopsy was performed in 92% of the patients, reflecting a tendency to biopsy very minor abnormalities. Because of this practice of biopsying minimal

Table 1. Presenting Signs and Symptoms of the 201 Patients in the Study at Mary Imogene Bassett Hospital

Sign/Symptom	Number of Patients (% of Total)
Cough	123 (61%)
Increased sputum production	92 (46%)
Weight loss	79 (39%)
Hemoptysis	60 (30%)
Anorexia	43 (21%)
Fever	40 (20%)
Dyspnea	23 (11%)
Pain	8 (4%)
Hoarseness	4 (2%)

irregularities, only slightly more than half (55%) of all biopsies provided a positive diagnosis; 59% of transbronchial biopsies were positive. Of positive biopsies, carcinoma accounted for 80%; the remaining 20% yielded other identifiable diagnoses (eg, sarcoidosis, lymphoma, *Pneumocystis carinii* pneumonia, tuberculosis). Of those patients who presented with the classic triad seen in lung cancer (hemoptysis, weight loss, and positive chest radiograph), 76% (13 of 17) were found to have a malignancy.

The most common complication associated with bronchoscopy at our institution (Table 2) was fever; of the 216 procedures in our study, 19 (about 9%) were complicated by fever of greater than 38°C within 48 hours. In addition to fever, we noted that four procedures (2%) resulted in hoarseness, two procedures (1%) resulted in hemoptysis, and one procedure (0.5%) in pneumothorax. There were no cases of bronchospasm, laryngospasm, myocardial infarction, or death.

We compared the expense of utilizing one respiratory therapist to assist a physician in bronchoscopy to the theoretical expense of bronchoscopy in the operating-room setting by determining the two costs to our institution. Because at our hospital all physicians are full-time employees, the cost of bronchoscopy outside the operating room was the sum of salary and fringe-benefit costs for the physician and respiratory therapist; costs for medication and equipment (ultrasonic nebulizer, flexible fiberoptic bronchoscope, biopsy forceps, disposable ultrasonic nebulizer circuit, and syringes); and a facilities charge for the fluoroscopic suite. The costs for a bronchoscopy in the operating room would be the sum of salary and fringe-benefit costs for the physician, certified registered nurse anesthetist, operating room nurse, operating room technician, and recovery room nurse; costs for medication and equipment, such as the bronchoscope and biopsy forceps; and facilities charge for the operating room and recovery room.

In an effort to clarify the comparison, we have expressed these costs per 10-minute unit of time. The costs to the institution for respiratory therapist-assisted bronchoscopy was $30.00. The theoretical cost to the institution for operating-room bronchoscopy was $88.40.

Discussion

Since the development of the flexible fiberoptic bronchoscope was reported by Ikeda in 1966,[3] the device has played a vital part in the assessment of the airway system. There is little dispute among physicians using this bronchoscope that its develop-

Table 2. Six Complications following Bronchoscopy: A Comparison of Results at Mary Imogene Bassett Hospital (MIBH) and Results of Four Previous Studies

Study (Total Procedures)	Number of Procedures with Complications (% of Total Procedures)					
	Pneumothorax	Hemorrhage	Respiratory Arrest	Death	Fever	Airway Obstruction
MIBH (216)	1 (0.5%)	2 (0.9%)	0	0	19 (8.8%)	0
Iowa[5] (600)	1 (0.2%)	6 (1.0%)	NR	0	NR	NR
Brompton[10] (1,223)	2 (0.2%)	7 (0.6%)	2 (0.2%)	0	2 (0.2%)	2 (0.2%)
Boston VA[17] (908)	6 (0.7%)	6 (0.7%)	2 (0.2%)	1 (0.1%)	11 (1.2%)	4 (0.4%)
Boston Univ[19] (100)	NR	NR	NR	NR	16 (16.0%)	NR

NR = not reported.

ment has been one of the most significant advances in pulmonary medicine.[1,4,5] Bronchoscopy has gained its popularity largely because of its versatility, effectiveness, and, above all, its safety.

The bronchoscope can be used not only for diagnosing endobronchial and parenchymal lesions, but also for obtaining culture material for bacterial, fungal, and viral studies. Therapeutically, this versatile device can be used to remove foreign objects and retained secretions, to direct laser therapy,[6] and to facilitate intubation. Research applications include the harvest of alveolar macrophages with the use of bronchial-alveolar lavage fluid,[7] tracheal mucus velocity studies,[8] and staging of lung cancer.[9]

We know that bronchoscopy is effective. Studies have shown that greater than 90% of bronchoscopic biopsies of directly visible lesions are positive and that 60% of transbronchial biopsies are positive.[5,10,11] Although transbronchial biopsy is not the procedure of choice for the diagnosis of interstitial pneumonitis,[12] it can be extremely helpful in other types of diffuse pulmonary disease, both infectious and noninfectious. Transbronchial biopsy has been found to be diagnostic in up to 76% of immunocompromised hosts.[13] A recent study found that in patients with acquired immunodeficiency syndrome (AIDS), transbronchial biopsy had a diagnostic rate of 79% for *Pneumocystis carinii* pneumonia.[14] In patients with sarcoidosis, transbronchial biopsy has been found to have a diagnostic rate as high as 90%.[15]

Regarding the factor of safety, further review of the literature shows that the overall complication rate of bronchoscopy is extremely low, with morbidity less than 1% and mortality even lower, with a death rate attributed to bronchoscopy procedures of only 0.08%.[4,5,17,18]

To analyze the results of a respiratory therapist's performing the biopsy under physician direction, we compared our rate of positive biopsies with the rates reported in the literature. Our results are statistically similar to or superior to those reported in the literature for both visible and nonvisible lesions (Table 3). As illustrated in Table 2, our rates of both major and minor complications are similar to those reported by other groups. The most troublesome complication for us was fever; however, the three largest studies in Table 2 either did not define fever or gave no indication of having looked for this complication in a systematic fashion. In the prospective study by Pereira et al at Boston University, fever was defined as being 38.5°C within 72 hours following bronchoscopy, and it was documented in 16% of 100 procedures.[19] These results are statistically similar to what we found in our own work ($P = 0.17$). The incidence of airway obstruction from laryngospasm or bronchospasm in our study also was similar to that in other studies.

The use of respiratory therapists as assistants in bronchoscopy has allowed us to offer quality service at a comparatively low price. Net-cost calculations show a substantial savings when use of the operating room is avoided. Our achieving results comparable to the national norms argues that we have utilized appropriate skill levels. We further believe that our results can easily be duplicated by other groups, provided their respiratory therapy staffs are motivated

Table 3. Bronchoscopic Biopsy of Tumor: Comparison of Results at Mary Imogene Bassett Hospital (MIBH) and Results of Three Previous Studies

	MIBH	Iowa[5]	Brompton[10]	Detroit[11]
No. of Patients in Study	201	600	1223	228
Visible-Tumor				
No. of patients biopsied	46	193	587	69
No. of positive biopsies	44	182	534	59
Rate of positive biopsies	95.7%	94.3%	91%	85.5%
Distal-Tumor				
No. of patients biopsied	64	137	75	15
No. of positive biopsies	38	97	48	7
Rate of positive biopsies	59.4%	70.8%	64%	46.7%

and eager to learn new skills and aspects of health care. Finally, depending on the Medicare/Medicaid case mix under the prospective reimbursement mechanism, institutional costs may well be significantly less if our approach is used.

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Safe and Successful Delivery-Room Intubation and Resuscitation of Meconium-Stained Newborns by Respiratory Therapists

Robert Perlson RRT

Prompt suctioning of the airways of meconium-stained infants at delivery reduces the incidence of disease associated with meconium-stained fluid (MSF). At Rogue Valley Medical Center in Medford, Oregon, trained respiratory therapists identify such infants needing intubation and perform the intubation and resuscitation in the delivery room. This study analyzed the effects of resuscitation by respiratory therapists who were called to the deliveries of infants with suspected MSF over a 5-month period. Method: I examined charts to tabulate data on meconium consistency (particulate or watery); whether births were vaginal or cesarean; whether intubation was performed and, if so, whether by a therapist or physician; and number of intubations per patient. I also tabulated outcome in terms of incidence of meconium aspiration syndrome (MAS), admission to the NICU, laryngeal complications of intubation, pulmonary air leak, and survival. Results: In the course of 700 consecutive births, 100 calls were made for respiratory therapists to attend deliveries of MSF babies; 74 babies were intubated, 69 by therapists. All 74 babies survived, and no laryngeal complications related to intubation occurred. Of the MSF babies whom therapists decided did not require intubation, none suffered subsequent respiratory distress. The rate of MAS was 1%, which is higher than that of another group of investigators who performed intrapartum oropharyngeal suctioning on all MSF babies; consequently, we are revising our protocol to place more emphasis on oropharyngeal suctioning and less on direct tracheal suctioning. Conclusion: I believe that the results of this study support the hypothesis that well-trained respiratory therapists can safely and successfully perform delivery-room resuscitation procedures involving newborns with meconium-stained fluid. (Respir Care 1986;31:689-694).

Introduction

In 1974 Gregory et al reported that amniotic fluid was stained with meconium in approximately 1 of every 11 births and that in about 25% of these cases the meconium was described as thick or like "pea soup."[1] An asphyxial insult to the fetus, causing relaxation of the anal sphincter, may precipitate the passage of meconium and subsequent staining of the amniotic fluid, or such staining may result from prenatal defecation, particularly in post-term fetuses.[2-4] In the study by Gregory et al, aspirated meconium was present below the vocal cords in 58% of the babies born through MSF who did not receive intrapartum suctioning.[1] This may represent an emergency because the aspirated meconium can cause chemical pneumonitis and/or complete or partial airway obstruction,[5] with a high incidence of persistent pulmonary hypertension[6] and pulmonary air leak,[1] events collectively known as meconium aspiration syndrome (MAS).

Mr Perlson is Director of Rehabilitation, Rogue Valley Medical Center, Medford, Oregon. He was Assistant Director, Department of Respiratory Therapy, at the time of the study.

Several investigators have demonstrated that prompt suctioning of the airways of meconium-stained infants at the time of delivery significantly reduces disease associated with MSF.[1,7-9] Studies using animal models confirm the theoretical basis for the delivery-room approach to the prevention of MAS.[10] Nevertheless, no study has been reported that clearly quantifies either the incidence of MAS in untreated MSF babies or to what degree mortality or morbidity is affected by suctioning MSF babies in the delivery room. Epidemiological studies before the advent of modern resuscitation and the specialized respiratory care of newborns showed that the mortality associated with thick meconium plus fetal distress was 21-32%.[2,3] More recent reports on symptomatic MAS showed death rates ranging from 6% to 12% for meconium-stained infants admitted to a neonatal intensive care unit (NICU).[7,11] Part of the difficulty in attributing an incidence to MAS is that it is often associated with perinatal asphyxia,[6] which remains a significant cause of morbidity and mortality even after the effects of meconium aspiration are eliminated. Ting and Brady reported that one group of babies born through MSF who received no tracheal suction at birth had a respiratory distress rate of 57%, with a 25% mortality rate, but that a comparable group of babies who were suctioned after birth had a respiratory distress rate of 28% and a mortality rate of only 1%.[7] However, they did not clearly define "respiratory distress." There is also evidence that those babies who do die after aspirating meconium are in a distinct group that is predisposed to persistent pulmonary hypertension by anatomical disorders that occur well before birth.[12]

Widespread acceptance of delivery-room treatment of babies born through MSF started with the publication of the 1974 paper by Gregory et al, who recommended that "all infants born through thick, particulate, or 'pea soup' meconium should have their trachea intubated and aspirated immediately after birth by the most experienced person in the delivery room . . . ."[1] More recently, the emphasis has been shifted to intrapartum suctioning of the neonate's oropharynx in the following manner, as outlined in 1976 by Carson et al:[9]

1. As soon as the baby's head appears on the perineum, the obstetrician passes a DeLee suction catheter through the nares to the level of the nasopharynx and aspirates any mucus or meconium, then suctions the mouth and hypopharynx.
2. Immediately after delivery, the pediatrician suctions the oropharynx with a bulb and, if meconium is present, inspects the cords by direct laryngoscopy while an assistant monitors the heart rate.
3. If meconium is present at the cords, direct suctioning of the trachea with either a DeLee catheter or oral endotracheal tube is performed.
4. Usual measures of ventilation and resuscitation are carried out.

Carson's group implied that immediate direct tracheal suctioning is warranted if the intrapartum suctioning is omitted or improperly performed.

Although the previously cited reports state that direct tracheal suctioning, when performed, should be repeated until the airways are clear, they are not explicit as to whether other resuscitative measures, such as positive-pressure ventilation, are to be applied between suction passes.

In all the published reports of which I am aware, resuscitation to prevent MAS was performed by physicians even though the standards of the American Academy of Pediatrics state that "responsibility for identification and resuscitation of the distressed infant may rest with a physician or may be delegated to appropriate trained . . . respiratory therapists."[13] Respiratory therapists have performed this duty at Rogue Valley Medical Center (RVMC) for approximately 10 years. To analyze the effect of resuscitation by respiratory therapists in terms of patient safety as well as the prevention of MAS, I undertook this study of patient outcomes after meconium-stained births at RVMC.

Materials and Methods

Training Program

Our training program for certification of respiratory therapists in neonatal intubation consists of the following steps:

1. The therapist-trainee attends an intubation workshop at which, following a presentation of didactic concepts, the trainee performs intubation techniques on kittens, as outlined by Kisling and Schreiner.[14]

2. The trainee observes intubation/resuscitation procedures performed by an experienced resuscitator at a minimum of three deliveries.
3. The trainee performs intubation/resuscitation procedures, under the close supervision of an experienced respiratory therapist, at at least three deliveries.
4. The trainee works on the "buddy system" for several months so that a more experienced therapist will be available for backup support during resuscitation in the event that problems arise.
5. The trainee is periodically recertified in an intubation workshop by the medical director of the NICU.

Protocol

Our delivery-room protocol is similar to that of Carson et al,[9] with the following modifications:

1. Respiratory therapists are called to attend all high-risk deliveries, including those through MSF. A physician resuscitator does not usually attend a vaginal delivery; rather, the respiratory therapist is usually assisted by a delivery-room nurse when resuscitation is required. Both a respiratory therapist and physician resuscitator attend a delivery by cesarean section.
2. The obstetric practitioner decides whether DeLee suction is to be used; not all meconium-stained babies at RVMC are suctioned with a DeLee device.
3. Direct tracheal suction via endotracheal tube is also indicated by the following situations, which are listed in the respiratory therapy protocol: (a) if there is thick meconium, (b) if there are clinical signs of airway obstruction; and (c) "if there is a question as to the need to intubate."
4. If necessary, oxygen and/or bag-mask ventilation is administered after the first intubation and suction, and suctioning is then repeated as indicated.
5. To prevent meconium from getting into the resuscitator's mouth during suctioning, a 'meconium trap' (Fig. 1) is attached to the end of the endotracheal tube.

Study Methods

I examined respiratory therapy charting on all calls in a 5-month period for respiratory therapists to attend deliveries of babies with suspected MSF, and I tabulated data on the consistency of the meconium (particulate or watery), as judged by the therapist; the route of delivery; whether intubation was performed and, if so, whether by a respiratory therapist or a physician; and the number of intubations per patient.

I then examined the charting for outcome in terms of symptomatic MAS, admission to the NICU, laryngeal complications of intubation, pulmonary air leak, and survival. Because the symptoms of asphyxia and MAS are often similar, a baby with "symptomatic MAS" is here defined as one who (1) was born through MSF, (2) had a chest radiograph consistent with aspiration pneumonitis or pulmonary air leak, and (3) had a higher-than-normal oxygen requirement (based on a PaO_2 of less than 55 torr while breathing room air).

Results

Of 700 babies born during the 5-month study period, respiratory therapists were called to attend 100 deliveries (14.3%) in which MSF was suspected;

Fig. 1. A 'meconium trap' constructed of a nebulizer (Bird Products Corp, Palm Springs, California) with a disposable mouthpiece at one end and a gauze baffle inserted in the middle.

74 of the 100 babies (74%) were intubated. Table 1 presents data on route of delivery, meconium consistency, and intubator.

The consistency of the meconium was the most important factor in the intubation decision; the intubation rate was 97% for those born with particulate meconium and 65% for those born with watery meconium.

Respiratory therapists intubated 69 (93%) of the babies undergoing intubation in this series; two of those 69 also received intubation passes by a physician. At the 11 cesarean-section deliveries where intubation was performed, respiratory therapists passed the tube 64% of the time, in spite of the presence of a physician resuscitator.

Table 1. Incidence of Meconium-Stained Fluid (MSF), MSF-Associated Intubation, and Particulate Meconium: Results of a 5-Month Study of 700 Births at Rogue Valley Medical Center

Suspected-MSF* Babies		
No. born in study period		
Total	100	(14%)*
By cesarean section	15	
Vaginally	85	
No. intubated		
Total	74	(74%)*
Cesarean-section deliveries	11	
Vaginal deliveries	63	
No. intubated by physician	5	(7%)
No. intubated by therapist	69	(93%)†
Cesarean deliveries	7/11	(64%)
Vaginal deliveries	62/63	(98%)
No. born through		
Particulate meconium	30/100	(30%)
No. intubated	29/30	(97%)
Watery meconium	55/100	(55%)
No. intubated	36/55	(65%)
Meconium of unreported consistency	15/100	(15%)
No. intubated	9/15	(60%)

*At delivery some babies suspected of having MSF actually had unstained or very lightly stained amniotic fluid. Thus, this study's MSF incidence rate (14%) overstates, and its MSF-associated intubation rate (74%) understates, the true respective rates.

†Two of the 69 babies also underwent intubation passes by a physician.

In 69 instances, the number of intubations required to clear the airway was reported:

No. of Passes Required	No. of Babies
1	26 (35%)
2	20 (27%)
3	13 (17%)
4	4 (5%)
5	3 (4%)
6	2 (2%)
7	1 (1%)
?	5 (7%)

One baby met the three criteria for MAS after being treated in the delivery room. That infant, the daughter of a diabetic mother, was born without a heartbeat and required full cardiopulmonary resuscitation after the first intubation/suction pass. She was managed on a ventilator for 2 days and was discharged home after 11 days. Her neurodevelopmental assessment at 1 year of age showed no deficits.

Four other babies from this series were admitted to the NICU. Three were treated for asphyxia or hyperviscosity syndrome, and one was treated for a pneumothorax but did not require supplemental oxygen.

No babies developed laryngeal complications related to delivery-room procedures, and no babies died. Of the MSF babies whom the respiratory therapists chose not to intubate, none suffered subsequent respiratory distress.

Discussion

This paper describes a program of delivery-room treatment for meconium-stained newborns and demonstrates that respiratory therapists can successfully fill the role of resuscitator for that condition. In this group of patients, the mortality and morbidity associated with meconium aspiration were minimized, consistent with the published results of others.[1,8,9] No harm to patients was documented as attributable to the meconium-aspiration protocol.

The 14.3% incidence rate of MSF in this study overstates the true rate, as some of the 100 babies suspected of having MSF had unstained or very lightly stained amniotic fluid at birth. For the same reason, the 74% MSF-associated intubation rate understates the true rate.

This study did reveal that the respiratory therapists

who attended these resuscitations were aggressive in performing endotracheal intubations, as evidenced by the high rate of intubation. Carson et al[9] needed to intubate less than 1% of their study sample after careful intrapartum oropharyngeal suctioning with a DeLee trap. The lack of emphasis placed on intrapartum suctioning at RVMC plus the phrase in our protocol that encourages intubation "if there is a question as to the need" may be responsible for our high rate of intubation. Because of this finding, our protocol and educational focus are being revised to put more emphasis on intrapartum oropharyngeal suctioning and less emphasis on direct tracheal suctioning.

Table 2 compares aspects of this study with those of three other published reports in which respiratory therapists had not been involved. Although outcomes (incidence of pneumothorax, laryngeal complications, symptomatic MAS, and survival) in our program at RVMC compare well to outcomes in the physician-based programs, the comparisons must be made in only a general way, as conditions and definitions were not strictly matched among studies. For example, the populations in the comparison studies were dissimilar to the population seen at RVMC, and prenatal monitoring and treatment have changed significantly in the years since the other studies were published. Furthermore, intrapartum oropharyngeal suctioning by obstetric practitioners was not part of either of the earliest two studies. In the series by Carson et al, who did utilize intrapartum suctioning, only one baby among 273 births was symptomatic for MAS.[9]

Nonphysician personnel have long been utilized to perform resuscitation and emergency intubation. However, I believe that the delegation of delivery-room resuscitation to respiratory therapists is relatively uncommon. A telephone survey of seven western United States hospitals that have NICUs revealed that in only two were respiratory therapists resuscitating meconium-stained newborns. If this current lack of therapist involvement is also true in community hospitals with obstetric services, respiratory therapists may have an opportunity to contribute significantly to a decrease in neonatal mortality and disease.

In summary, these data support the hypothesis that a team of well-trained respiratory therapists can safely and successfully perform delivery-room intubation of meconium-stained newborns.

Table 2. Oropharyngeal-Suction Application, Intubation Rates, Incidence of Thick Meconium, and Outcome in Four Studies of Meconium-Stained-Fluid Births

	Rogue Valley Medical Center	Carson et al[9]	Fox et al[8]	Gregory et al[1]
Intrapartum oropharyngeal suction?	Yes, on some babies	Yes, on all babies	No	No
Percentage of MSF babies intubated	74%*	< 1%	100%	91%
Incidence of "thick" meconium	30%	Not reported	31%	24%
Incidence of pulmonary air leak	1%	Not reported	8.6%	8.8%†
Incidence of laryngeal complications	0	Not reported	0	0
Incidence of symptomatic MAS‡	1%	<1%	20%	18%
Survival rate	100%	100%	100%	100%

*Some of the 100 "MSF" babies in this study actually had unstained or very lightly stained amniotic fluid at birth; thus, an intubation rate of 74% for MSF babies understates the true rate.

†All babies received chest radiograph study; only 2.5% were symptomatic.

‡Defined as necessitating supplemental oxygen administration and producing a chest radiograph consistent with aspiration pneumonitis and/or pulmonary air leak.

ACKNOWLEDGMENTS

I am grateful for the assistance in data retrieval rendered by the staffs of the Medical Records and Quality Assurance Departments at Rogue Valley Medical Center, to Dr William Fox of The Children's Hospital of Philadelphia for his encouragement, and to Dr Bonnie Lees at RVMC for her most helpful criticism.

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Respiratory Care Devices and Techniques through the Years

Dean Hess MEd RRT

Technology probably plays a greater role in respiratory care than in any other health profession that involves direct patient care. I have often commented to my students that respiratory care practitioners must be combination physiologists, pathologists, and technologists. We have to understand how our patients function in health (physiology) and in disease (pathology). Further, we need to know how patient-care equipment works (technology). Most importantly, we must be able to adapt respiratory care devices to the needs of the patient (eg, adjust ventilator flow to meet patient demand), we must recognize when a device is not functioning correctly (eg, a leak in a ventilator circuit), and we must recognize a patient's adverse response to a change in technology (eg, a drop in blood pressure following the establishment of PEEP).

For as long as I have been a respiratory therapist, we have been expected to cleverly adapt equipment to meet patients' needs. I well remember placing the expiratory tube from a ventilator into a bucket of water to produce PEEP, placing a Bird exhalation valve backwards in the inspiratory limb of an MA-1 circuit to produce IMV, and using a needle splint to patch the severed pilot tube of an endotracheal tube cuff. Few respiratory care practitioners who joined the profession in the current microprocesser era appreciate the pleasures of disassembling (and better yet, reassembling) the guts of an Emerson Post-Op ventilator (could I still do it?), or cleaning the Bennett valve from a PR-1 ventilator, or taking apart and putting together a Bird Mark 8 (anybody remember NEEP?).

My favorite recollection of a respiratory therapist's technologic improvisation comes from the mid-1970s. A colleague of mine needed to set up CPAP (which was a relatively new thing at the time), but no anesthesia bags were available; they were all fulfilling their intended use—ventilating patients in the OR. So this clever therapist went to the pulmonary function lab, got an old leaky weather balloon, and patched its holes with adhesive tape. He then dumped some 10-cc syringes out of their carton and stuffed the weather balloon into the empty carton to produce a semi-compliant CPAP reservoir of the appropriate volume. Guess what? It looked terrible, but it worked well! Like many others, the patient got better because of the ingenuity of a respiratory therapist.

It was with pleasure that I accepted the invitation to write this commentary to accompany the 11 papers that follow. Because I was in the First Grade in 1956 (when Volume 1 of INHALATION THERAPY appeared), I feel that I kind of grew up with the Journal (although I did not know of its existence before the early 1970s). The past 35 years of INHALATION THERAPY and RESPIRATORY CARE are an excellent portrayal of the evolution of respiratory care technology, as is shown by the 11 papers republished in the following pages, which date from 1956 to 1983.

Some False Starts

The first three articles come from the early days of INHALATION THERAPY, and they represent some 'false starts.'

Schwartz and Dale: **Addition of Dead Space To Produce Hyperventilation for Prophylaxis of Atelectasis** (1956)—The opening sentence of this paper is as true today as 35 years ago: "Atelectasis is recognized as a common and often dangerous complication, occurring not only after surgery but also in other situations in which tracheobronchial secretions are not properly cleared." The only thing

Mr Hess is Assistant Director of Clinical Research, York Hospital, and Instructor, School of Respiratory Therapy, York Hospital and York College of Pennsylvania—York, Pennsylvania.

that seems to have changed over the years is the 'vogue' treatment for atelectasis at any given moment. The hyperventilation therapy reported by Schwartz and Dale never became very popular, unlike IPPB in the 1960s and early 1970s, and incentive spirometry from the mid-1970s to the present. I found it interesting that in 1956 Schwartz and Dale thought it necessary to define P_{CO_2} and P_{O_2}. Other differences from modern papers are that the authors did not describe the equipment with which they made their measurements, there was no statistical analysis of the data, and no references from the literature were cited.

Siegman: **Oxygen Tents Go Streamlined at Johns Hopkins** (1961)—This paper describes a "streamlined" oxygen tent that is actually a face tent. It is interesting that the options for oxygen administration in 1961 were a mask, a nasal catheter, or a tent. It is difficult for me to imagine the practice of respiratory care without the nasal cannula. In spite of the now-common use of the nasal cannula, the face tent still has some limited use today, mainly in patients with facial trauma. This paper too listed no references, and it described methods of investigation but gave no data.

Wehner: **What Is Electro-Aerosol Therapy?** (1963)—The title is appropriate. Evidently not many respiratory care practitioners knew what electro-aerosol therapy was in 1963, and probably fewer know today. Perhaps we can't even state that this therapy had a false start, as I don't believe it was ever accepted by the health-care community in America. This paper also provides no supporting data for its claims.

Toward a Firmer Foundation

The remaining eight articles have more to do with respiratory care as we practice it today.

Glick and Benner: **Arterial Oxygen Tension during Oxygen Breathing** (1968)—Here is an important paper from the early days of the respiratory care discipline. First, Glick and Benner showed that the nasal cannula was nearly as effective as the nasal catheter for oxygen administration. Second, they showed that the nasal cannula can be effectively used even if the patient breathes through the mouth, which disposed of an old bias against cannulas. Interestingly, the study involved the use of an 18-gauge arterial catheter, but there is no mention of informed consent or the approval of an institutional review committee; the research environment has definitely changed in this respect since 1968.

This paper's authors are of personal interest to me, as well. Victor Glick was the director of the respiratory therapy department and school at St Joseph Hospital in Lancaster, Pennsylvania—which is where I was born and where I attended inhalation therapy school. John Benner directed the inhalation therapy department at another hospital in Lancaster. Both Glick and Benner are now physicians, and Dr Glick is an Associate of this journal.

Kittredge: **IPPB—The Pressure Is Building** (editorial) (1973)—This editorial is a very fitting selection in this group of papers intended to illustrate the technology through the years. No one has done more for our Journal than Phil Kittredge, Editor for two decades and now serving as Adjunct Editor. This 1973 editorial is characteristic of the challenge that Phil has issued to practitioners for many years—to be professional, to be scientific, to be part of the solution rather than part of the problem, and to "be all that we can be." Phil Kittredge has probably had a greater impact on my career for the past 10 years than any other person, and I suspect that many others could say the same. Although his editorial on IPPB was written more than 15 years ago, several of its points are as true today as then. For example: "Some of these therapists know better. Some really do not. A certain number used to give better therapy but haven't the time anymore. Some have simply lost their spirit. Many have put an end to the formerly overt conflict by telling themselves, 'It doesn't matter about the bad therapy because most of the patients don't need it anyway.'" He also wrote, "At present, our field is perhaps 90 per cent artful guesswork and only 10 per cent science." I wonder how much that ratio has changed since 1973.

Benson and Pierson: **Ventilator Wash-Out Volume: A Consideration in Endotracheal Suction Preoxygenation** (1979)—The first point I want to make about this paper is that it was written by a therapist (Benson) and a physician (Pierson). In my opinion, we need more therapists teaming with their medical directors and other physicians to perform research, to analyze unusual cases, and to write papers together to be submitted to RESPIRATORY CARE. My

second point is that this paper deals with an everyday, practical matter. I suspect that many times therapists want to tackle 'un-doable,' esoteric research projects, while important projects like this one remain not done. My third point is that this work could have been done in any respiratory care department, large or small. It did not use any special equipment, nor did it require a large research budget. Essentially, all it took was the time and dedication of a therapist-physician team.

Scacci: **Air-Entrainment Masks: Jet Mixing Is How They Work; The Bernoulli and Venturi Principles Are How They Don't** (1979)—Sometimes we think we understand something well, only to learn that we had it all wrong! Such has been the case with air-entrainment masks, as Dr Scacci told us in this journal in 1979. Just when we had become comfortable calling them "venturi" masks, we learned from him that these devices do not work in the ways that we had been taught. Since this paper appeared in RESPIRATORY CARE, I have often wondered whether we have misunderstood any other principles, passing fiction along from generation to generation of students and practitioners. Anything else up your sleeve, Dr Scacci? I also wonder how many naive clinicians still refer to "venturi" masks.

Estey: **Subjective Effects of Dry Oxygen versus Humidified Low Flow Oxygen** (1980)—Another big surprise for many of us. No sooner had Scacci unmasked "venturi" masks than along came another heretic, Mr Estey, to suggest that not all supplemental oxygen has to be humidified. Really? In respiratory therapy school, we learned that in normal breathing the inspired gas is almost fully humidified by the time it reaches the lower respiratory tract—and that medical oxygen is stored as a dry gas. So it made sense that we should humidify the oxygen we deliver to our patients. However, this reasoning failed to account for a few things: (1) Commercially available humidifiers for low-flow oxygen are not very efficient, (2) the upper respiratory tract is normally a very efficient humidifier of inspired air, and (3) when low-flow oxygen is breathed, most of the inspired gas is from the ambient air, not from the oxygen supply. The Journal had been around 25 years before Mr Estey dropped his bombshell, and the noise wasn't heard in many places for some time, if ever. But as a result of his work and other reports, many hospitals no longer use humidifiers with low-flow oxygen. The cost savings to society from the elimination of humidifiers with this therapy are tremendous. This article shows that an important publication need not be lengthy—this one is only two pages long!

Demers, Pratter, and Irwin: **Use of the Concept of Ventilator Compliance in the Determination of Static Total Compliance** (1981)—Some things are commonly known, but not commonly made use of. I think that one of them is the concept of compressible volume in ventilator circuits. Because the effect of ventilator compliance is often small in adult ventilation, many clinicians fail to recognize its effects when it is important, such as in the presence of low tidal volumes and high ventilating pressures. Demers et al have nicely illustrated the consequences of failing to consider ventilator compliance and PEEP when we calculate total compliance. Although the effect of PEEP on the calculation of compliance is now widely appreciated, I suspect that the effect of ventilator compliance on that calculation is still not fully understood, even though this paper appeared almost a decade ago. Like the Benson-Pierson paper of 1979 and Estey's 1980 report, this paper by Demers et al shows that 'old' literature is not necessarily out of date. A later development related to Demers et al's message is the concept of auto-PEEP as another influence on the calculation of static compliance; this too is frequently not considered.

Beckham and Mishoe: **Sound Levels inside Incubators and Oxygen Hoods Used with Nebulizers and Humidifiers** (1982)—The lay public commonly considers the hospital to be a "quiet zone." However, we who work in them know that often this is not the case. As Beckham and Mishoe discovered, nebulizers and humidifiers can produce sound levels high enough to be potentially harmful to the development of infants' hearing. I believe that's just the beginning of noise hazards in hospitals. The noisy ICU may be the site of sleep deprivation and anxiety for patients. I have often wondered how noise may interfere with the concentration of hospital personnel. For example, the noise in the report room in many respiratory care departments (and nursing stations) can be deafening at the change of shift. One can only speculate on the effect this may have on patient care: wrong information passed on, missed treatments, medication errors, and the like.

Off, Braun, Tompkins, and Bush: **Efficacy of the**

Minimal Leak Technique of Cuff Inflation in Maintaining Proper Intracuff Pressures in Patients with Cuffed Artificial Airways (1983)—In my opinion, the use of high-volume, low-pressure cuffs and attention to the volume and pressure of air put into airway cuffs have had an important effect on airway-related complications. It is my impression that major cuff-related injuries to the airway are much less common than they were 10-15 years ago. Much of this is the result of the careful attention that respiratory care practitioners pay to using correct cuff-inflation techniques and the maintenance of safe cuff pressures. Off et al reminded us of the problems and offered important guidance.

A Final Note

These 11 papers are only a small sample of the many useful and important reports on devices and techniques that have appeared in the Journal over its first 35 years. Initially I planned to offer a bibliography of those that couldn't be reprinted here. However, so many articles and editorials from former years carry vital bits of our knowledge base and technique storehouse that room forbids listing them. I encourage all readers to get to a library and dig out old issues of RESPIRATORY CARE and perhaps INHALATION THERAPY. You who have come into the field in the past 5 years or so have missed a lot, but it's been saved for you in the Journal's pages. Many of longer tenure in the discipline would enjoy reliving part of their professional past by making the same trip. Find an afternoon this winter to sit by the fire and read through the highlights of the past 35 years. Take it from me, it can be fun.

Cartoon reprinted from Respir Care 1978;23:1034.

"Sorry about your machine . . . I think I huffed and I puffed a little too much."

Reprinted from INHALATION THERAPY
[Inhal Ther 1956;1(2):16-18]

Addition of Dead Space to Produce

HYPERVENTILATION

for Prophylaxis of Atelectasis

By SEYMOUR I. SCHWARTZ, M.D. and W. ANDREW DALE, M.D., F.A.C.S.

ATELECTASIS is recognized as a common and often dangerous complication, occurring not only after surgery but also in other situations in which tracheobronchial secretions are not properly cleared. The prophylactic effect of hyperventilation induced by carbon dioxide breathing has been known for many years. The use of a paper bag into which the patient rebreathes and accumulates CO_2 is a common method of stimulating greater tidal volumes. However, the small size of the bags ordinarily used, as well as the constant leaks due to imperfect fitting about the face, makes this method inadequate. Rebreathing with 5 or 10% CO_2 mixtures, using a face contour mask, effectively increases aeration of the lung, but entails bulky apparatus and increased cost.

Recently we have achieved hyperventilation by simply extending the patient's normal respiratory dead space with a rubber tube. This results in an increased alveolar pCO_2*, and subsequently an increased *arterial* pCO_2, which by central nervous system stimulation causes hyperventilation.

Studies were performed on normal personnel in order to select a standard dead space tube. The average tidal respiratory volume in these subjects was 590 cc, and test runs demonstrated an average increase of 770 cc in the tidal volume when breathing through the added dead space. This resulted in a "stimulated tidal volume" of 1360 cc if a 1000 cc dead space were used. A 1000 cc tube, made of black rubber with an internal diameter of 3.2 cm and a length of 125 cm was therefore arbitrarily selected for patient use, because it more than doubled the control tidal volume. (See fig. 1) With the nostrils occluded either by the fingers or by a nose clip, the patients respired through this tube, using a Collins rubber mouthpiece connected to the tube by a metal joint.

Fourteen patients who underwent various surgical procedures were studied. Control tidal volumes and "stimulated tidal volumes" (with the addition of the 1000 cc dead space) were measured. Readings were taken pre-operatively and on the first three postoperative days. In all fourteen patients, the addition of the 1000 cc dead space approximately doubled the control volume preoperatively, and more than doubled it on the three postoperative days. There was no significant alteration of the respiratory *rate*. Tidal volumes, alveolar oxygen and carbon dioxide partial pressures were measured during a 5 minute control period and during 10 minutes of breathing through the added dead space.

These measurements were plotted on a graph, and the pCO_2 and tidal volume curves closely paralleled each other (see fig. 2). The greatest increment of "stimulated

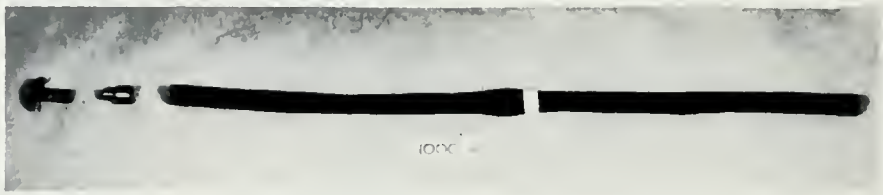


Figure 1. 1000 cc. rubber "added dead space" tube with rubber mouthpiece and metal connection.

*the symbol pCO_2 (or pO_2) is read, "partial pressure of CO_2 (or O_2)"

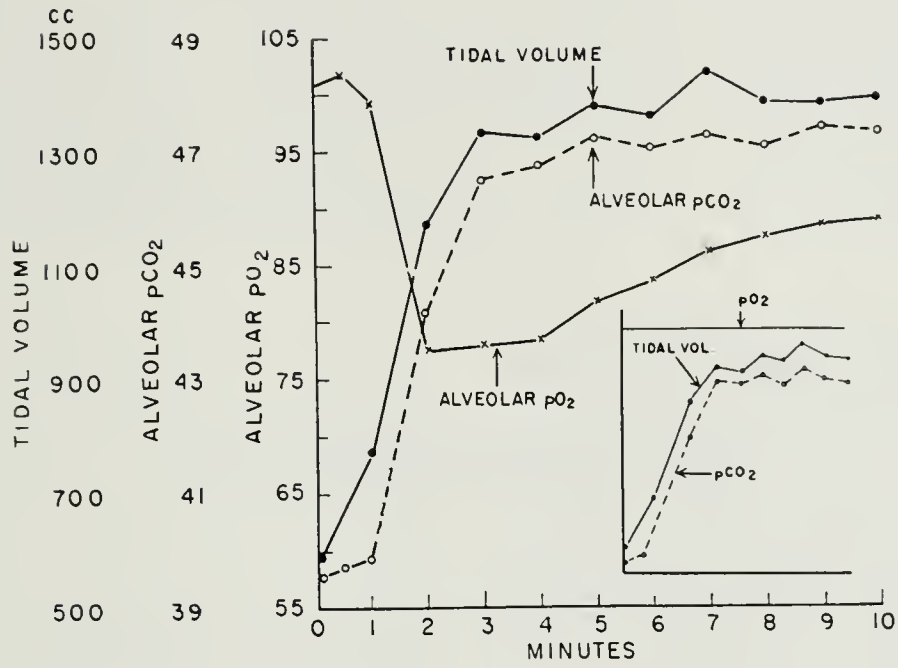


Figure 2. Average of 15 normal subjects breathing room air through the 1000 cc. "added dead space" tube showing the relationship between tidal volume, alveolar pCO_2 and alveolar pO_2. Note the decrease in alveolar pO_2 when breathing room air (main graph) and the maintenance of normal alveolar pO_2 in 3 subjects when 3 to 4 liters of oxygen per minute were introduced into the distal end of the tube (insert).

tidal volume" occurred during the first two minutes, during which time it increased from a control value of 596 cc to 1188 cc. During the third minute, there was a smaller but nevertheless significant increase in tidal to 1344 cc. From the third minute through the tenth minute, there was little change in the "stimulated tidal volume." Similarly, the greatest rise in alveolar pCO_2, from the control value of 39.44 mm mercury to 44.17 mm, occurred during the first two minutes. A second increase, from 44.17 to 47.20 mm mercury, was noted between the second and fifth minutes, and for the remainder of the ten minutes, the alveolar pCO_2 remained stable.

The partial pressure of *Oxygen* in the alveoli sharply diminished from the control value of 100.5 mm mercury to 77.61 mm during the first two minutes of hyperventilation, and over the remaining eight minutes steadily and gradually rose to 88.93 mm. If a flow of 3 to 4 liters of oxygen per minute was introduced into the distal end of the breathing tube, the average increased tidal volume and pCO_2 values were similar to the changes noted in the previous study. However, in this situation, pO_2 was maintained *above* the control value of 100.5 mm mercury. This indicates that while breathing through the 1000 cc of extra dead space, the elevation of the pCO_2 is the critical factor causing increased tidal volume.

Hyperventilation, with its attending pulmonary distension, has been considered an important factor in prevention of atelectasis. An increase in alveolar pCO_2 raises arterial pCO_2, which by central nervous system stimulation causes hyperventilation. The addition of 1000 cc dead space to the respiratory passage causes a significant rise in pCO_2 in the alveoli, and therefore effectively increases the respiratory volume.

Rebreathing the patient with such dead space tubes is presently being used throughout the Strong Memorial and Rochester Municipal hospitals in a program directed toward the prevention of atelectasis. A five minute rebreathing period every one to five hours has

been initiated, and subjectively patients have found this method easier than either the paper bag or 5% CO_2 via face mask. While the nurse pinches the patient's nostrils shut, he needs only keep the rubber mouthpiece in his mouth and no additional cooperation is required.

The tidal volume is doubled by the end of the three minutes of rebreathing, equilibrates at this point and then remains stable. The patient therefore is actually rebreathing a "stimulated tidal volume" of two times his unstimulated volume during the last two minutes of this period. The marked decrease in alveolar pO_2, which rises again after two minutes, is ordinarily well tolerated by the patient. In those individuals in whom anoxia is a concern, a flow of 3 to 4 liters of oxygen per minute can be directed into the open end of the rebreathing tube, resulting in the maintenance of a normal pO_2 while hyperventilation is being accomplished. This constitutes another distinct advantage over the paper bag method, in which there is constantly decreasing alveolar pO_2.

LOOKING AHEAD

An excerpt from the opening address delivered at the 1955 Institute by the Chairman of the Board of Directors, Sister M. Rudolpha, O.S.F.

I AM happy, once again, to preside at the opening luncheon of the Institute sponsored by the American Association of Inhalation Therapists.

I am happy that so many of you were able to come to this Institute. It is my hope that we shall continue to hold these annual meetings, and that because of them our influence will increase, and we shall earn not only the respect but the admiration of doctors, hospital administrators, and other professional people.

We have only begun to scratch the surface. When we consider that we have fewer than four hundred members, and that our potential membership can be estimated in the thousands—then we can readily see how much remains to be accomplished.

I have a picture in my mind for the future. Perhaps it is only a dream, but who can tell when dreams become a reality?

I see our association with chapters in principal cities all over the United States and Canada.

I see our membership in the thousands.

I visualize the respect and admiration with which others will regard us.

I see a Journal published regularly by our association, one very much in demand by all persons interested in inhalation therapy.

Other parts of this dream include a complete and competent bibliography, a permanent library containing the best that has been published on inhalation therapy.

Perhaps we shall even get to the place where we have licensing in all states, with uniformly high standards established with the help of our association.

And, finally, I see industry looking to us with respect and with a great degree of satisfaction because we are a thoroughly self-supporting, and completely professional association.

These are just some of the dreams for the future of the American Association of Inhalation Therapists. Won't you please join me and the other members of the Board in working hard to help these dreams come true?

Remember the ANNUAL MEETING

The 1956 Annual meeting of the American Association of Inhalation Therapists will be held in New York City at the Park Shereton Hotel, November 12-16. Plan to attend.

Reprinted from INHALATION THERAPY
[Inhal Ther 1961;6(2):16-17]

Oxygen Tents Go Streamlined At Johns Hopkins

by William L. Siegman
Chief Inhalation Therapist
Johns Hopkins Hospital, Baltimore, Maryland

OLDER oxygen administration methods have given ground to the inexpensive open-top face tent at Johns Hopkins. In 1959, with an average of 30 patients receiving oxygen simultaneously in the hospital, sixteen received oxygen through open-top face tents, nine by refrigerated tents, three by catheter, and two by face mask. The current trend in favor of the open-top face tent is primarily based on its operating simplicity, ability to maintain adequate concentrations, patient comfort, and low maintenance costs.

Because of its simplicity, the face tent requires practically no maintenance outside of routine cleaning after use. After being used, face tents are brought into the oxygen therapy department and checked for damage. At this time the damaged units are discarded. The face tents and tubing are washed and placed in an antiseptic solution for sterilization. After air drying, they are put in a glass-door cabinet in the oxygen therapy department for storage. Proper care has made it possible to use each open-top face tent several times before it is discarded.

The open-top design of the face tent provides a significant safety factor because even if the gas flow is blocked, atmospheric oxygen will be available to the patient. In addition, the face tent eliminates concern about the possibility of oxygen toxicity from the inhalation of high concentrations of oxygen, or the occurrence of apnea in the patient with CO_2 narcosis whose remaining hypoxic drive for respiration is removed by inhalating oxygen of too high concentration.

A number of patients will tolerate neither mask nor nasal catheter, and make it necessary to tie up expensive electric tents which are not actually indicated. In such a case, the patient will tolerate a face tent with very little difficulty. It has also been observed that some patients who are extremely apprehensive tend to exhibit less anxiety with a face tent than with the larger and better-known oxygen tent.

The use of the face tent has not been restricted to any particular type of patient; however, it is used most frequently on post-operative cases. The face tent is particularly useful on those patients who require a large degree of nursing care; frequent pulse and blood pressure determinations and the need for intravenous feeding or medication, etc., drastically reduce the concentration within a canopy, which must be opened and closed to provide the necessary service.

The post operative patient often requires the insertion of a Levine tube in one nostril. The use of nasal oxygen will cause both nostrils to be occupied, and the drainage tube will interfere with alternating the nasal catheter between nostrils to relieve nasopharyngeal irritation. In these instances, face tents allow the patient to receive the proper amount of oxygen regardless of nursing care, and with a minimum of discomfort. As the patient's condition changes, it may be desirable to utilize some other type of apparatus.

The face tent, currently available from many suppliers, fits comfortably over the lower portion of the patient's face and is open wide at the forehead to allow the escape of exhaled gases and vapors. Constructed from flexible, clear plastic, the unit is supported by an adjustable plastic headstrap. Its weight is only 1¼ ounces. It can be easily removed for washing and feeding, and constant attendance or expert supervision is unnecessary. Simultaneous Wangensteen suction drainage or Miller-Abbott tube feeding can be performed where indicated.

Humidified oxygen enters the fact tent through a shower-head dispersal system that eliminates many of the discomforts present with nasal catheters and face masks. A continuous flow of oxygen replaces the consumed gas, and CO_2 and warmed vapors flow out readily through the open top of the face tent. The flow of atmospheric air through the open top along with the oxygen entering the mask provide adequate dilution of CO_2 to a safe level.

Breathing, as well as visibility, is completely free and unobstructed at all times. Rarely has a complaint been made of claustrophobia or unpleasant odor.

"This photo shows the face tent being used with large bore tubing as an aerosol mask. When used for oxygen with standard humidification, the smaller input nipple is used."—Photo courtesy NCG

Reprinted from INHALATION THERAPY
[Inhal Ther 1963;8(6):18-25]

What Is Electro-Aerosol Therapy?

by Alfred P. Wehner, D.M.D.

ELECTRO-AEROSOL therapy (E.A.T.) is a special form of inhalation therapy characterized by high unipolar electric charges imposed on aerosol droplets. E.A.T. has been employed for some 20 years in Germany and is now administered in inhalation clinics in 14 other countries. The fact that German health insurance companies are recognizing E.A.T. attests to its acceptance by the medical profession in the country of its origin where more than 200,000 patients have received this treatment to date. Good results are claimed particularly in respiratory diseases, especially in bronchial asthma, bronchitis, hay fever, whooping cough, pulmonary emphysema, silicosis, and sinus conditions.[1] This article intends to give a brief review of E.A.T., which is all but unknown in this country.

Doctor Wehner is a Senior Research Scientist for the Biometrics Instrument Corporation, Dallas North Research Park, Plano, Texas

Development of Electro-Aerosol Therapy

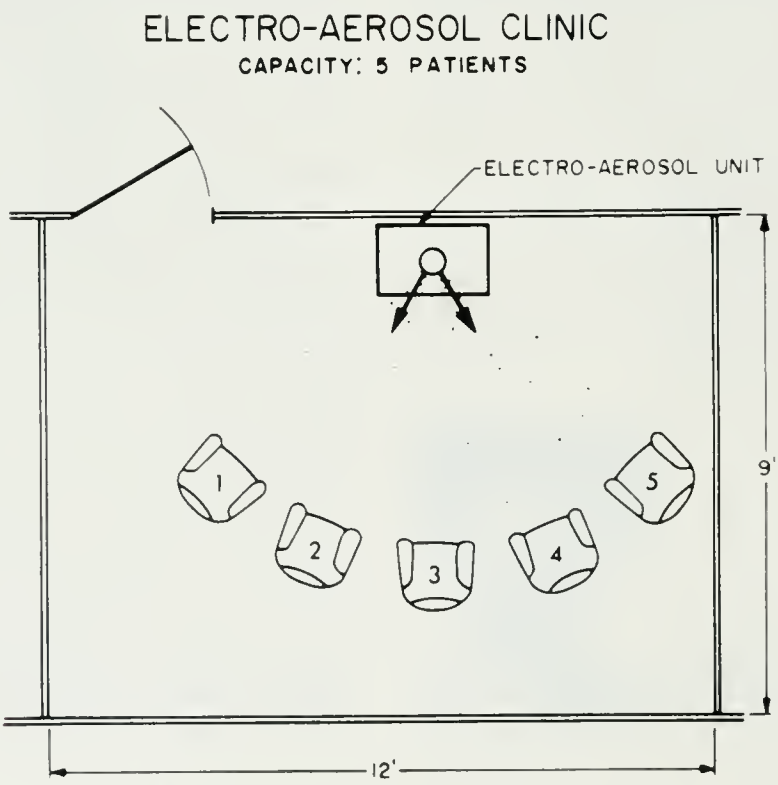
Observations on the effect of weather and climate on man can be traced back to the beginning of the medical literature. The role of meteorological factors in modern medicine is evidenced by the fact that a recent issue of the International Journal of Biometeorology lists 359 publications on this subject in the German professional literature from 1946 to 1962.[2]

Many of the biological phenomena believed to be caused by meteorological factors could not be explained satisfactorily in terms of temperature, relative humidity, and atmospheric pressure. Around the turn of the century increased attention focussed on a then new meteorological factor, the so called air ion. Air ions are electric charges attached to submicroscopic particles or clusters of gas molecules such as oxygen or carbon dioxide. Ions are continuously generated and die at the same rate. In higher altitudes cosmic radiation is the main source of air ionization. At lower levels radioactive substances in the earth are responsible for the atmospheric ion density. The ratio of positive and negative ions and perhaps the rate of change of this ratio appear to be biologically effective agents. When increasing evidence indicated beneficial effects of negative and detrimental effects of positive ions the

application of artificially ionized air in medicine suggested itself.

This has, indeed, been attempted with various degrees of success. The classic work of Dessauer in Germany and the studies of Tshijevsky in the Soviet Union heralded this new era in inhalation therapy.[3,4] Advanced work in this field is now being conducted at several research centers in the United States[5] and, to a much larger extent, in the Soviet Union. But why not combine the effect of electric charges with the well established beneficial effect of conventional inhalation therapy as applied, for instance, with vaporizers? This consideration led to the development of what is now known as Electro-Aerosol Therapy.

E.A.T. is thus a predominantly empirical therapy. This means that its application is based on past experience in a large number of patients and on clinical cause-effect relationship, but its mode of action is still poorly understood. In other words, it is pretty well known WHAT electro-aerosols do in respiratory patients, but HOW they do it is still the subject of considerable research effort. This holds true, though, for various other forms of treatments and many drugs at that. While this situation is in many instances acceptable to the practitioner whose main interest lies in the effective relief of discomfort and pains, it is unsatisfactory to the scientist whose objective it is to find an explanation of the effective mechanism. However, encouraging progress has been made toward this goal during the past 10 years. Effects of inhaled electric charges on the central nervous system, blood pressure, blood differential count, pH, sedimentation rate, BMR, vital capacity, and various other physiological parameters have been investigated. An effect on hormone and enzyme activity has been independently found by several investigators. A discussion of this work would exceed the scope and purpose of this article. It may suffice to say that more scientifically acceptable data on this subject exist than most sceptics are aware of (reviewed in references 1 and 5).



Schematic Diagram of Small Electro-Aerosol Clinic (Top view)

Technical Description of Electro-Aerosol Systems

There are several methods by which electro-aerosols can be produced. One system employs a set of centrifugally arranged jets mounted in the center of a bowl shaped open glass vessel. The wall of the vessel acts as baffle plate for the aerosol. Upon impingement on the baffle plate the aerosol droplets break up into finer droplets. The larger ones fall back into a water reservoir, the finer ones rise and leave the system. While leaving they are directed by a mushroom shaped guide toward the inhaling patients. The guide is located centrally above the glass vessel and the jet. A set of tungsten wires extends from the periphery of the mushroom top down to the upper edge of the glass bowl. The wires are maintained at a potential of 40 to 60 kilovolts and reportedly impart a charge of 4 to 1000 elementary units (1 elementary unit = 1.59×10^{-19} coulombs) to each droplet leaving the system.[6] A compressor provides the required air pressure for the dispersion of the aerosol.

Another system uses ultrasonic energy with frequencies between 1 and 6 megacycles for the dispersion of the aerosol. Ultrasonically generated aerosols are characterized by finer droplets, better homogeneity, and higher aerosol density than other dispersion aerosols. Its proponents claim that these advantages compensate for the reduced charge carrying ability of electro-aerosols of ultrasonic origin.[7] It appears, though, that this method has not advanced beyond its experimental status of some nine years ago.

Another method was developed by Dr. Küster, professor at the Goethe University Medical School, Frankfurt, Germany, and Barthel, an electrical engineer, 20 years ago. To date improved versions of this system are apparently the most successful and best known form of E.A.T.

There are two modifications of this system available. The large unit is designed for group therapy in special inhalation clinics in hospitals, spas, mining companies and china factories (stone dust!), and similar in size and shape to a home type refrigerator. It is placed in the center of a 20 ft. x 20 ft. x 11 ft. inhalation room. The jet and liquid reservoir are mounted on top of the assembly (Figures 1-3). The Barthel jet is a metal sphere with eight jet openings for the radial dispersion of the aerosol. The liquid phase enters the center of the sphere from the liquid reservoir by means of a regulated gravity drive. The liquid is led to the periphery of the sphere through eight small channels. A compressor provides an air pressure of 50 p.s.i. at the channels for the atomization of the liquid. The mean droplet diameter of the aerosol can be regulated by inserting a calibrated pin of a selected size in the channel. The diameter determines the site of deposition, that is the depth of penetration of the droplets, in the respiratory tract. The therapeutic effect of an inhaled aerosol is a function of the droplet size, all other parameters remaining unchanged. Droplet diameters between 0.5 and 5 microns achieve maximum penetration and deposition of the aerosol in the depth of the lung.

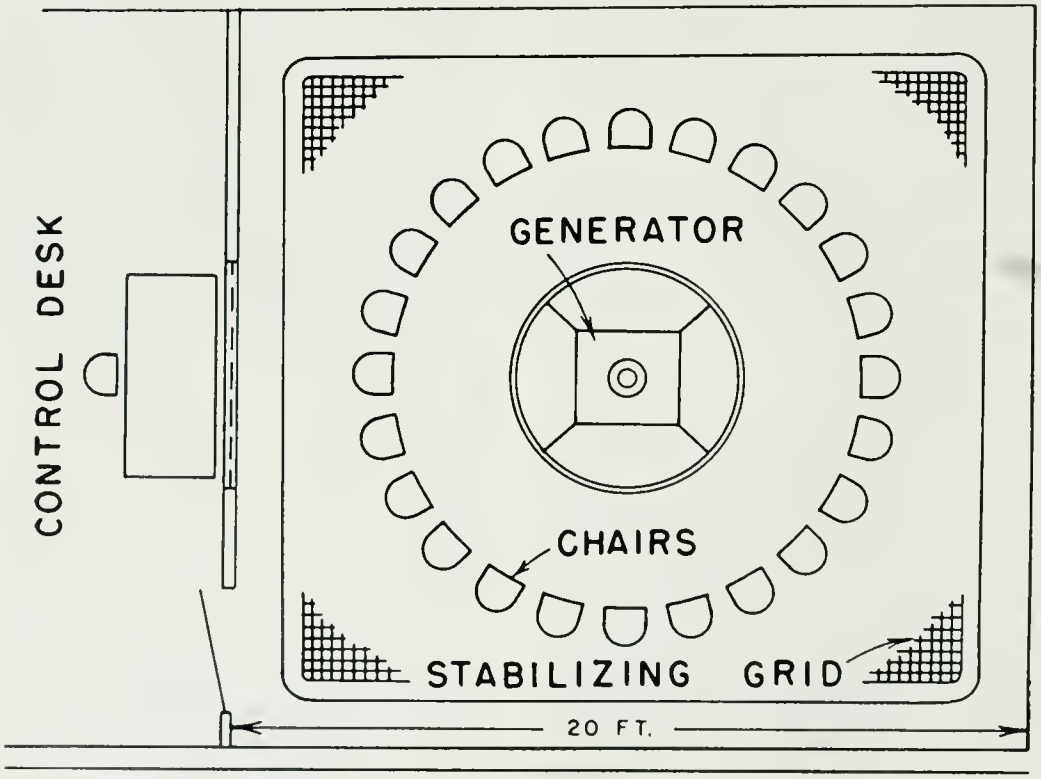
A high voltage power supply maintains the jet at either a negative or positive potential of 40 to 60 kv. The electrical current is limited to 120 microamperes thus eliminating any safety hazards. This arrangement imparts a charge of up to 1600 elementary units to each individual aerosol droplet.[8] The "unipolarity" of the electro-aerosol is greater than 90 per cent. In addition to the therapeutic effect, the charges enhance deposition of the aerosol in the lung.[9-13] Furthermore, the mutually repelling force of unipolar charges reduces the number of collisions with subsequent coalescense and rapid sedimentation of the droplets. Electro-aerosols are therefore more homogeneous and more stable than comparable neutral aerosols. Up to 20 patients can be treated simultaneously with the large unit. They are placed around the unit at a distance of approximately 5 feet from the jet. Good grounding of the patient is important to prevent a build up of an electrostatic potential which would repel the electro-aerosol. Suspended from insulators under the ceiling is a stabilizing grid carrying the same charge and polarity as the jet. The grid improves electrostatic field conditions and

concentrates the electro-aerosol in the area occupied by the patients.

Some investigators and clinicians, among them the Russians, use plain or distilled water as the liquid phase. Most other investigators, including myself, prefer to use slightly hypertonic saline solutions. The liquid phase is apparently not a particularly critical factor unless a medicated solution for a specific therapy is used. E.A.T. with medicated solutions, by the way, appears to be a promising combination whose therapeutic potential should be further investigated. Bronchodilators, detergents, and mucolytic agents can be used for a non-specific inhalation therapy; antiphlogistics, antibiotics, tuberculostatics and others may be used for a specific therapy. This mode of drug application circumvents the gastro-intestinal system. It eliminates the necessity of flooding the entire organism with a drug in order to obtain the required concentration at the affected locale, which may result in undesirable side effects.

The small Küster-Barthel unit delivers an electro-aerosol of similar characteristics to that produced by the larger unit. The aerosol is charged by a potential of 27 kv and is dispersed through two jets diverging at an angle of 60 degrees. Up to 5 patients can be treated simultaneously. They are seated in a semicircle about the unit approximately 5 feet from the jet. The small unit does not require special facilities for installation. It can be placed in the corner of an ordinary room with minimum dimensions of 100 square feet which makes it well suited for use in small clinics or private offices (Figures 4 and 5).

The technical supervision of the electro-aerosol inhalatorium is usually delegated to an inhalation therapist. The operation of an electro-aerosol system does not require special technical knowledge or special skills once it has been properly installed. However, the success of the therapy depends to a large extent on a competent technical supervision governed by a few basic rules.



Schematic Diagram of an Electro-Aerosol Inhalatorium (Top view)

Clinical Aspects And Treatment Procedures

The electro-aerosol treatment is a rather pleasant experience for the patient. No inhalation masks are required, thereby eliminating such problems as sterilization, maintenance and replacement as well as potential difficulties with problem children and infants. Due to their physical properties, electro-aerosols are non-wetting and do not produce either fog or uncomfortably high humidity. The patient may read, doze or otherwise relax. Breathing instructions during the treatment by a breathing simulator in the form of a video and audio presentation to the patient may be employed to enhance the effect of the inhalation therapy.

Chronic cases require generally a minimum of 20 daily treatments of 45 minutes duration. Some patients require 30 or more sessions. Acute cases are treated until the symptoms subside. In some cases rather dramatic results during the first treatment can be observed.

Patients with asthma, bronchitis, pertussis, hay fever, and pneumoconiosis appear to be most responsive to E.A.T. Most patients are reported to show clinical improvement and various degrees of subjective relief or even complete remission of their symptoms. The level of improvement is usually maintained considerably beyond the duration of the treatment in spite of the fact that patients often return to an unfavorable environment conducive to relapses. The viscosity of mucus is markedly reduced leading to easy expectoration of mucus and finally, in many cases, to the cessation of wheezing and coughing. Patients breathe better, the typical exasperating night cough and asthma attacks disappear, appetite increases and the general well being improves. Failures are comparatively rare. Yet it would be naive to expect unrealistic results. A chronic condition like asthma or emphysema which has entrenched itself in the organism over a period of years is not likely to be relieved over night. Sometimes a relapse may be precipitated by a meteorological front or by a newly acquired disease such as a severe cold. In other patients the effect of E.A.T. may wear off after a period of time. In these cases an after treatment of 10-15 sessions is indicated. In many cases we cannot hope for more than a temporary subjective relief, particularly when irreversible changes of the pulmonary tissue have occurred. The observed subjective and clinical improvement in these cases is presumably due to liquefaction and expectoration of mucus resulting in a more efficient operation of the remaining functional tissue, and possibly to a favorable effect on the autonomic system.

Large Küster-Barthel type electro-aerosol inhalatorium in operation (Courtesy Elektro-Aerosol Ziems, Cologne, Germany)

Clinical observations and investigations by authorities on E.A.T. do indeed suggest a direct effect of electro-aerosols on the autonomic system [14-17], which regulates the involuntary body functions such as blood pressure, defensive actions in case of a disease, and many others. The interdependence of the autonomic system and somatic and psychic events is well known. A localized somatic condition has its effect on the functions of the autonomic system. Vice versa, a derangement of autonomic functions may result in various somatic manifestations. Any therapy improving the condition of this important center tends to have beneficial secondary effects on these somatic manifestations.

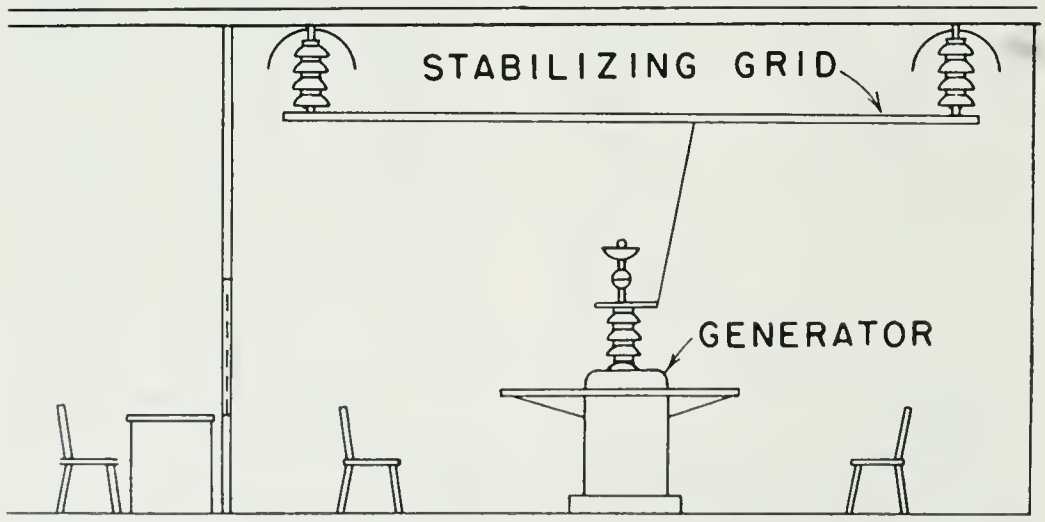
The central effect of E.A.T. on the autonomic system could explain satisfactorily the frequently observed sustained symptomatic relief. A merely local effect of the electro-aerosol on the irritated respiratory tissue, for instance, could at best account for a temporary improvement. Any subsequent irritation of the organism in the allergic phase would lead to a relapse. A restoration of the autonomic function, however, will enable the organism to tolerate these irritants by the proper physiological reactions of the autonomic system.[15] A central effect of electro-aerosols can also account for results in other than respiratory diseases where a local effect is precluded. The observed increased resistance of patients under electro-aerosol therapy to noxious agents (Pathogenic microorganisms, allergens, etc.) can also be plausibly explained by a "tune-up" of the autonomic system, resulting in top efficiency of the organism's defensive mechanism.

There is no reliable method to date to determine the required polarity of the electro-aerosol for the individual patient prior to the treatment. Reaction of the patient to the treatment has to serve as guidance until a satisfactory method and sufficiently sensitive instruments have been developed. Empirism suggests to start patients on negatively charged aerosols. Only those who show lack of results are put on positively charged electro-aerosols.

Summarizing this review it can be said that, based on the available evidence, E.A.T. appears to be a meritorious adjuvant in the treatment of respiratory diseases when judiciously applied by a qualified operator.

Electro-Aerosol Therapy In The United States

E.A.T. in the United States is still in its infancy. We are, for good reasons, reluctant to adopt any therapy that has not been thoroughly tested by our own medical schools, hospitals, and research institutes. The first steps in this direction have already been taken. A Veterans Administration Hospital near Dallas, Texas, recently started a research program investigating the efficacy of E.A.T. in pulmonary emphysema. Measurements with a Keithley electrometer (Model 200A) and a 1000:1 voltage divider (Model 2007) with an input impedance of 10^{12} ohms showed a space charge potential of 8000 volts due to the electrically charged aerosol droplets, in the area occupied by the patients. Studies with radioactive tracer aerosols conducted at a U. S. Naval Medical Research Laboratory verified results of German investigators showing that the electric



Schematic Diagram of an Electro-Aerosol Inhalatorium (Cross section)

charge on the droplets significantly increases the penetration and deposition of aerosols in the lung. This by itself would justify the clinical application of electro-aerosols, particularly of medicated solutions as it is done, for instance, in German tuberculosis sanatoriums.

Another clinical research program commenced recently at the University of Southern California Medical School.

In addition, there are four small electro-aerosol systems of the Küster-Barthel type operating in private clinics. Preliminary clinical results with these small units in more than 200 patients are quite encouraging and definitely suggestive of beneficial effects of this therapy.[18] A representative case history from our own electro-aerosol clinic may be of interest. The 39 year old patient developed severe chronic asthma after the birth of her first child in 1954. Conventional therapeutic measures, including allergy injections over a period of three years, were ineffective. In January 1962 the patient received a series of electro-aerosol treatments with rather dramatic results. She remained asymptomatic and did not use any drugs for a period of 15 months. In May 1963 a severe cold provoked a relapse which required repeated hospitalization in the following months. In October the patient, being on antibiotics, bronchodilators and various other types of medication, reported for a new series of electro-aerosol treatments. The patient again responded very well and became asymptomatic after a few treatments. Claiming to feel excellent she is now no longer on medication. Similarly favorable results and case histories are on record. Beyond this apparent cause-effect relationship and clinical and subjective evidence, I have been able to demonstrate significant changes of the galvanic skin resistance of patients under electro-aerosol treatment which is indicative of an effect of the electro-aerosol on the autonomic nervous system. [15,19,20,21] Notwithstanding this apparent success I would like to emphasize that a scientifically valid evaluation of E.A.T. will have to be based on statistically significant and reproducible results from controlled studies in this country which are now under way.

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Arterial Oxygen Tension During Oxygen Breathing*

R. Victor Glick, ARIT, John N. Benner, ARIT*****

THE use of oxygen for the relief of hypoxia has been well established in clinical medicine.[1-3] Previous studies[4-7] have evaluated the effectiveness of oxygen therapy technics by measuring oxygen concentrations in inspired gas, nasopharyngeal gas, or alveolar gas.

The purpose of this study is to evaluate the effectiveness of oxygen therapy by measuring arterial oxygen tension while subjects breathe oxygen from three types of equipment in current clinical use. This study measures the concentration of oxygen delivered to patients receiving routine oxygen therapy.

Materials and Method

Oxygen tension of arterial blood taken from 12 healthy volunteers between the ages of 19 and 26, was studied while the volunteers breathed from three types of equipment. Each subject breathed oxygen from a nasal cannula, a nasal catheter, and a partial rebreathing mask. A B-D eighteen gauge teflon catheter was placed in the brachial artery of each subject under local anesthesia. Blood samples were collected anaerobically in heparinized syringes at 20-minute intervals as the subjects breathed oxygen from the different types of equipment. Samples were analyzed immediately for oxygen tension using the Clark type oxygen electrode manufactured by Radiometer of Copenhagen. The electrode was calibrated before each study using a solution with zero

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** Director School and Department of Respiratory Therapy, St. Joseph Hospital, Lancaster, Pa.

*** Chief Respiratory Therapist, Lancaster General Hospital, Lancaster, Pa.

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TABLE 1. *Range and Mean Oxygen Tension Using the Five Methods.*

	Range Pa_{O_2}	Mean Pa_{O_2}
Cannula Mouth Open	200-320	240
Cannula Mouth Closed	240-320	270
Catheter Mouth Open	200-310	260
Catheter Mouth Closed	210-330	270
Mask	310-450	385

oxygen tension and one equilibrated to room air. Standardization of the electrode with higher concentrations of oxygen was carried out intermittently, as necessary. Oxygen was delivered through an Ohio, standard adult, pressure compensated flowmeter and bubble jet humidifier.

The following procedure was established for each subject. Each subject was allowed to relax and quietly breathe room air for 10 minutes after the artery was cannulated. A blood sample was then taken and analyzed for pH, Pco_2, and Po_2. A determination of 38-42 mm. Hg, Pco_2, was considered normal and was used to show that normal minute ventilation had returned after arterial cannulation. Accepted standard oxygen flows were used for the various methods. Oxygen was then administered to each person via a nasal cannula—Hudson model number thirty—at a flow rate of eight liters per minute. Each subject was instructed to breathe through his mouth. After 8 to 10 minutes of oxygen breathing the second blood sample was taken and analyzed for oxygen tension. The oxygen was then turned off and the subject breathed room air for an 8 to 10-minute period. Oxygen was then started at the same flow rate of eight liters per minute and the subject was instructed to breathe with his mouth closed. After 8 to 10 minutes of oxygen breathing the third sample was taken and analyzed for oxygen tension. The same procedure was followed using a nasal catheter—Hudson size twelve French—at a flow rate of six liters per minute. Lastly, a Hudson plastic elongated partial rebreathing mask—model number 24—was used at a flow rate of eight liters per minute. Careful attention was given to insure all three types of equipment had been placed correctly. All catheters with the exception of one were visualized in the oral pharynx with the tip of the catheter placed posteriorly to the uvula. All subjects were free from apparent cardio-respiratory disease so that our arterial oxygen findings are representative of the efficiency of the different methods of oxygen delivery.

Results

The average oxygen tension while the subjects breathed room air was 104 with a range of 92-111 mm. Hg. Table 1 shows the Pa_{O_2} values using the five methods described.

If normal gas exchange is assumed then inspired oxygen concentration can be calculated from the Pa_{O_2} values in the following manner. The P_{AO_2} can be found by adding 10 mm. Hg (normal A-a gradient) to the Pa_{O_2}. The oxygen tension in inspired air (P_{IO_2}) is derived by adding 40 mm. Hg (P_{ACO_2}) to the P_{AO_2}. The F_{IO_2} can then be calculated by dividing the P_{IO_2} by 713.

Discussion

Arterial oxygen levels achieved by the oxygen mask were significantly higher than those obtained by the catheter or cannula. One reason for this difference was that the new elongated design of the mask allows for a tight fit on most of the subjects. The fit of the mask is important and was demonstrated by a lower arterial oxygen tension in one of our subjects with a poorly fitting mask. Another important reason for higher oxygen levels using the mask is because the partial rebreathing bag acts as a reservoir for oxygen. The mask produced significantly higher oxygen

levels in all cases except where the mask fit was poor. Oxygen delivered by either nasal cannula or nasal catheter, whether the subject breathes with his mouth open or mouth closed, will raise the arterial oxygen tension to levels which will essentially saturate the hemoglobin with oxygen. Because adequate arterial oxygen tension is achieved by either the catheter or cannula, regardless of whether the subject is a mouth or a nose breather, and because there is essentially no difference in cost, the choice of equipment should be based on patient comfort.

All subjects complained of nasopharyngeal irritation, difficulty in swallowing, and dryness of the throat using the nasal catheter. All subjects were eager to remove the catheter upon completion of the study. This reaction was in contrast to the complete lack of discomfort experienced by all subjects using the nasal cannula. It was obvious that the nasal cannula was far more comfortable than the nasal catheter.

It has been generally believed that the effectiveness of oxygen delivered by nasal cannula is reduced if the patient is a mouth breather. Kracum[7] has stated, "If the patient is a mouth breather, the concentration of oxygen obtained by either catheter or cannula will approximate that which is present in room air." Our results as well as the studies of Kory[4] do not uphold this statement. Our results show oxygen is delivered to the alveoli by nasal cannula even when the subjects breathe through their mouths. Oxygen delivered through the nasal cannula is apparently drawn into the inspired gas flow and this enriches the oxygen concentration.

Summary

Arterial oxygen tension was measured in 12 healthy subjects between the ages of 19 and 26 to evaluate the effectiveness of oxygen delivered by nasal cannula, nasal catheter and face mask. Highest oxygen levels were achieved using the mask. Oxygen levels did not vary significantly using nasal cannula or nasal catheter whether the subject breathed through his mouth or nose. Overall evaluation of comfort placed the cannula first, mask second, and catheter last.

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IPPB–The Pressure Is Building

The title of our lead article this month—**Why Are They Saying Bad Things About IPPB?**—may jolt a few hardy souls who have not known anyone was criticizing the sacred cow of respiratory therapy. But, like the fellow who was so dumb he didn't *know anything,* they may feel better upon learning that others are so dumb they don't even *suspect* anything.

Dr Ziment tells us in this article that IPPB is not a science, that it may not be any good, and that if it is good, we certainly are not sure what it's good for. But he did not touch on two very sensitive points: one point is that IPPB is Big Business; the other is the fact that so much IPPB is poorly given. Both give critics plenty to talk about, and neither seems to be discussed in public.

IPPB is big business because most hospital respiratory therapy departments are expected (or expect themselves) to be self-supporting and even to be profit-making, and many of them depend on IPPB for up to 75 per cent or more of their billings. Thus, the greater part of the respiratory therapy community in this country (perhaps 25,000 to 35,000 persons—no one really knows how many) derives its income and job security from IPPB. Some departments own 100 IPPB machines or more—an investment of some $50,000—as well as several thousand dollars worth of spare breathing circuits and backup parts. And they may spend several thousands of payroll dollars per month to see that those machines are used. That is big business.

When so much money is tied up in IPPB equipment and in salaries to pay staff members who were hired on the expectation of requests for IPPB therapy, then IPPB becomes a vested interest. The situation can easily blind the department manager to some of the emerging realities of contemporary patient care.

Although medical care delivery should be managed on a businesslike basis, it remains unethical to "advertise" for "business." Nevertheless, when a "business recession" hits a hospital (when not enough persons in the community are ill), it often brings consternation to the department manager, who sees his capital investment lying idle and his staff drawing pay while "income" falls. Some persons in this situation, being captive of their vested interests and being afraid that cracks are appearing in their empires, or at least that the hospital administrator will frown when he reads the monthly report, have been known to prowl the hospital halls for likely patients, buttonholing their doctor friends to ask, "Haven't you got anyone we can put on IPPB?"

Any "business" breaks even or shows a profit simply by keeping expenses below income. In a hospital department aiming to break even or better, one can reduce expenses for equipment and labor, or raise prices, or seek more revenue-producing procedures. Often the administration is loath to hire more labor, because that increases costs, and if the department is already making money they hope to maintain the status quo. Thus, although many technical directors fight to get enough staff members to meet the IPPB load adequately, they often fail. The result usually is that it is *impossible* to give IPPB patients their money's worth. There just are not enough labor hours available to meet the IPPB demand.

If one divides the number of IPPB "treatments" given in some hospitals by the number of real hours of IPPB labor (not time spent making rounds or setups or in coffee shops), it turns out that the average IPPB treatment lasts perhaps five minutes—or less. When you consider that *some* of the patients probably got good 15- or 20-minute treatments (and such a treatment takes 30 minutes of labor when you figure in preparation, cleanup, and charting time), then it follows that in order to create the average of five minutes per treatment, some of them must have lasted only a minute or two.

The fact is that we defraud many patients. And that is one way to put it when asking a hospital to hire more labor.

The corollary to this is that, most likely, relatively few IPPB treatments are given well. My observations over the last decade convince me that a great many therapists give IPPB treatments without knowing exactly what the aim of therapy is for any given patient. Most of those to whom I have talked do not usually coach the patient. Few see that the patient holds his breath at end-inspiration to facilitate gas and aerosol distribution. Few cough him after the IPPB. Some of these therapists know better. Some really do not. A certain number used to give better therapy but haven't the time anymore. Some have simply lost their spirit. Many have put an end to the formerly overt conflict by telling themselves, "It doesn't matter about the bad therapy, because most of the patients don't need it anyway."

When we reach that stage, we have begun to play doctor.

The fact that IPPB labor is not equal to the demand not only defrauds the patient but also makes it very difficult to study and assess the therapy. Dr Ziment makes much of the fact that no one has really proven that IPPB is beneficial, and that various observers differ in their conclusions. No doubt a great part of the problem is that it is hard to find good IPPB to study. Treatments in one location and at one time cannot validly be compared to those given elsewhere and at another time; they are too likely to be given entirely differently. And in a large sampling (which is what we need for a study), the odds are high that many treatments will be incompetent.

Perhaps IPPB is useful, or could be useful, but proof is difficult to come by when the procedure is not yet uniform or scientific. It may even be invalid to include in one study two treatments given the same patient on a single day, if one is given by one therapist in the daytime and the other is given by a second person in the evening. As likely as not, they march to different drummers. Thus, I doubt that any extensive studies of adequate IPPB have yet been made.

If one side of the coin shows that hospitals should hire more therapists to meet the IPPB load, the other side shows that physicians should order more selectively. But even when doctors have been educated about the supposed indications for IPPB, the practice of defensive medicine inclines many of them to "routine" ordering of IPPB, perhaps in ignorance that what the patient may get is "perfunctory" IPPB. There is little to be done about this until the literature shows these physicians what IPPB can and cannot do.

I once made chart rounds with my medical director to show him how IPPB was being unnecessarily ordered. In response, he mounted a vigorous campaign among the house staff to cut the routine orders, asking them, by way of challenge, if they ordered antibiotics on all their patients "in case they might get an infection." After that, IPPB orders *increased,* and my medical director said to me, "You know, doctors have a lot of divine rights."

Many therapists are defensive when the value of IPPB is questioned. They do not like to think that a $500 respirator, which they know inside and out, is not more valuable in some cases than a $3.75 rebreather, which they have never bothered to take apart and understand. Hell, the respirator *looks* more important, doesn't it?

And yet, given the impact of many contemporary IPPB treatments, it is likely that a patient would benefit more from having the therapist stand by the bed or chair and encourage deep breathing for 10 or 15 minutes.

By the nature of statistics, our group includes numbers of a recent generation that taught itself to trust *feelings* more than facts—to make their judgments on the basis of "good vibes" rather than good sense. To them it may be seductive to *feel* that IPPB must be good, especially if it en-

ables them to earn $125 to $175 a week after a bit of painless training.

It is time to stop feeling that "everything will work out." Instead we should ask: Are the patients being taken care of? Or are they being taken in?"

And let us consider the unpleasant question my medical director once asked me: "What are you going to do when IPPB goes out of fashion?"

The question is as fair as it is disturbing. We must remember that we are *respiratory* therapists, not inhalation therapists or IPPB therapists. We must help in the search for the answers about IPPB. And if it goes out of fashion, we must be ready to offer other skills to the patients. Respiratory disease will not disappear; it will always have to be treated. But if IPPB is to have an honored place in that treatment, we can help identify the role and then deliver the goods.

Dr Ziment's article concludes with the impetus for us to ask the right questions in the right places—our hospitals. He points out ways in which technical personnel can participate in the decisions about IPPB. At present, our field is perhaps 90 per cent artful guesswork and only 10 per cent science. But *we* can have a hand in turning those figures around, especially as we are closer to the patients than the doctors or the laboratory investigators. Particularly suitable for study by therapists and technicians are several of the 11 suggestions with which Dr Ziment closes his article. We encourage research by our readers and look forward to receiving papers based upon it.—*PK*

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[Respir Care 1979;24:832-835]

Ventilator Wash-Out Volume: A Consideration in Endotracheal Suction Preoxygenation

Michael S Benson BS RRT and David J Pierson MD

Mechanically ventilated patients requiring endotracheal suction are often preoxygenated with an inspired oxygen fraction (Ft_{O2}) of 1.0 for a brief period prior to the suction procedure. Because the "wash-out volume" required to displace an existing gas mixture with pure oxygen throughout the ventilator circuit has not previously been measured, we determined this volume for each of five commonly used ventilators. Using an oxygen analyzer at the distal end of the tubing circuit and varying tidal volume (VT), we found wash-out volumes ranging from 6.3 l for the Monaghan 225 ventilator at VT 700 ml to 18.0 l for both the Bennett MA-1 and Ohio 560 ventilators at VT 1,500 ml. The Bourns BEAR-1 and Servo 900-B ventilators gave intermediate volumes. Depending upon tidal volume and cycling frequency, a new Ft_{O2} may not actually be delivered to the patient for as long as 2 minutes after the ventilator controls are changed. Hypoxemia due to endotracheal suctioning may not be prevented if the ventilator is used for preoxygenation and allowance is not made for wash-out volume.

Introduction

Endotracheal suctioning procedures may cause hypoxemia,[1-4] which can be severe and lead to fatalities.[5] Measures recommended for prevention of hypoxemia include limiting the duration of suction,[1] providing supplemental oxygen,[2] and increasing ventilation.[3,6] For patients receiving mechanical ventilation the inspired oxygen fraction (Ft_{O2}) is often increased prior to,[1,4] during,[2,4,7] and for a period after the suctioning procedure. Preoxygenation, usually for several breaths before insertion of the suction catheter, may be carried out using an anesthesia bag, a self-inflating manual resuscitator with high-Ft_{O2} reservoir, or the ventilator itself. Because the latter method is often used in our intensive care unit, with several ventilator breaths being delivered after the oxygen control knob is turned to Ft_{O2} 1.0 prior to suctioning, we studied the time-volume course of preoxygenation with five widely used ventilators. The purpose of the study was to determine the "wash-out volume" of each ventilator, defined as the volume required to displace fully a given gas mixture from the ventilator and its tubing. For application to endotracheal suction preoxygenation, this would represent the volume necessary to achieve delivery of a measured Ft_{O2} of

Mr Benson is Team Coordinator, Respiratory Therapy Department, Harborview Medical Center; and Dr Pierson is Medical Director, Respiratory Therapy Department, Harborview Medical Center, and Assistant Professor of Medicine, University of Washington, Seattle, Washington.

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Reprint requests to Dr Pierson, 325 Ninth Ave, Seattle WA 98104.

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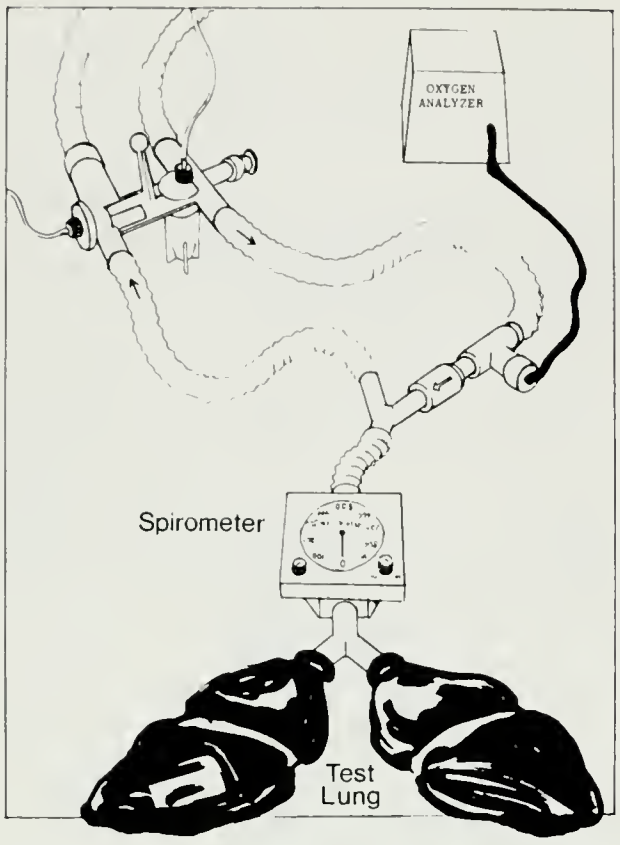

Fig. 1. Diagram of apparatus used in determining ventilator wash-out volume.

1.0 to the patient after the FI_{O2} control has been turned to an indicated 1.0, and it would be useful in determining how long one should wait before initiating suctioning.

Methods and Materials

The apparatus used in this study is diagrammed in Figure 1. Each ventilator was connected to two rubber test lungs by a standard manifold and disposable tubing (Bard Parker Co, Rutherford NJ). A fuel-cell type oxygen analyzer (Hudson Oxygen Therapy Sales, Temecula CA) with a response time of 15 seconds was placed in-line at the distal (patient) end of the inspiratory tubing. Oxygen readings were taken after each ventilator cycle. Because of the slow cycling frequency employed to allow equilibration of the oxygen analyzer between breaths, a one-way valve was inserted between the inspiratory and expiratory arms to eliminate retrograde flow from the test lungs into the oxygen analyzer. Ventilators used were the Bennett MA-1 (Bennett Corporation, Kansas City MO), the Ohio 560 (Ohio Medical Products, Madison WI), the Monaghan 225 (Monaghan Co, Littleton CO), the Bourns BEAR-1 (Bourns Inc, Riverside CA), and the Servo 900-B (Siemens Corporation, Union NJ). Tidal volumes were 700, 1,100, and 1,500 ml. A Cascade humidifier (Bennett Corporation, Kansas City MO) was placed in the ventilator circuit in each instance, and all measurements were made with the humidifier water level at "full." Volume and FI_{O2} were recorded with each ventilator cycle, and "wash-out volume" was calculated as the product of tidal volume and the number of ventilator cycles required to attain the desired FI_{O2}. Data were obtained in triplicate at each tidal volume for each ventilator used.

Results

Figure 2 illustrates the data generated in this study, and shows the wash-out curve for the Bennett MA-1 at tidal volume 1,100 ml and FI_{O2} 0.21 and 0.50. To reach a measured FI_{O2} of 1.0 required 16 cycles (17.6 l) at each starting FI_{O2}. At a rate of 9 cycles/min, approximately 2 minutes would be required before the actual delivered FI_{O2} matched that dialed on the ventilator. Starting with FI_{O2} 0.5, after

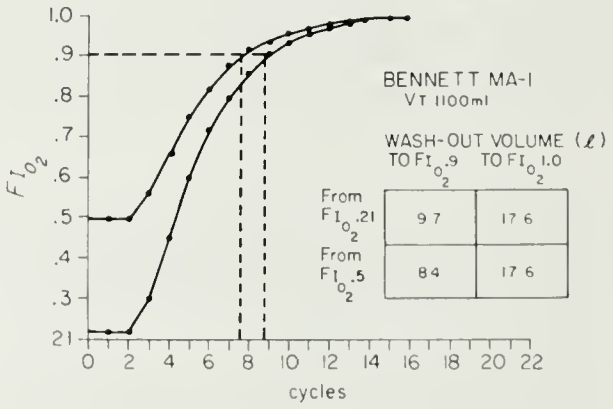

Fig. 2. Wash-out curve for the Bennett MA-1 volume-cycled ventilator. Wash-out volume is calculated as the product of tidal volume and the number of ventilator cycles required to reach the desired oxygen fraction (FI_{O2}).

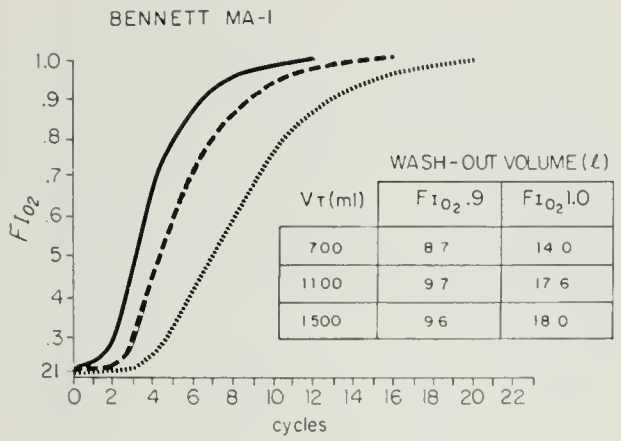

Fig. 3. Wash-out curves and calculated wash-out volumes to $F_{I_{O_2}}$ 0.9 and 1.0 for the Bennett MA-1 using V_T 700 ml (solid line), 1,100 ml (heavy dashed line), and 1,500 ml (fine dashed line).

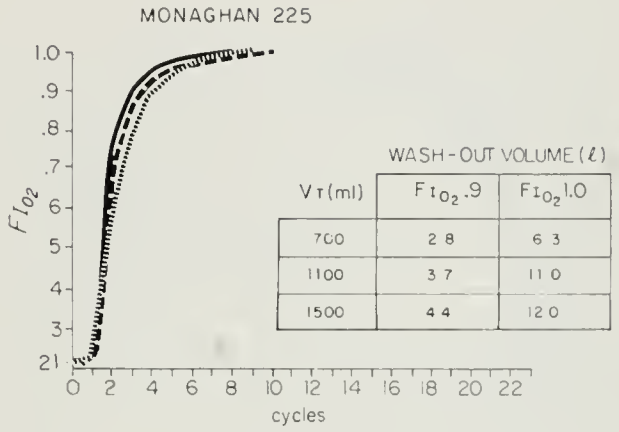

Fig. 5. Wash-out curves and calculated wash-out volumes to $F_{I_{O_2}}$ 0.9 and 1.0 for the Monaghan 225, using V_T 700 ml (solid line), 1,100 ml (heavy dashed line), and 1,500 ml (fine dashed line).

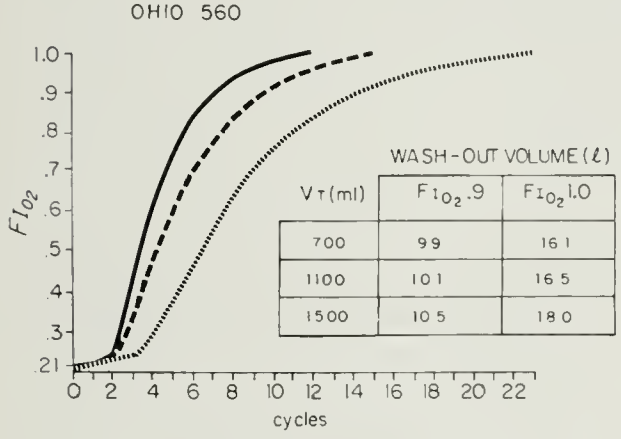

Fig. 4. Wash-out curves and calculated wash-out volumes to $F_{I_{O_2}}$ 0.9 and 1.0 for the Ohio 560, using V_T 700 ml (solid line), 1,100 ml (heavy dashed line), and 1,500 ml (fine dashed line).

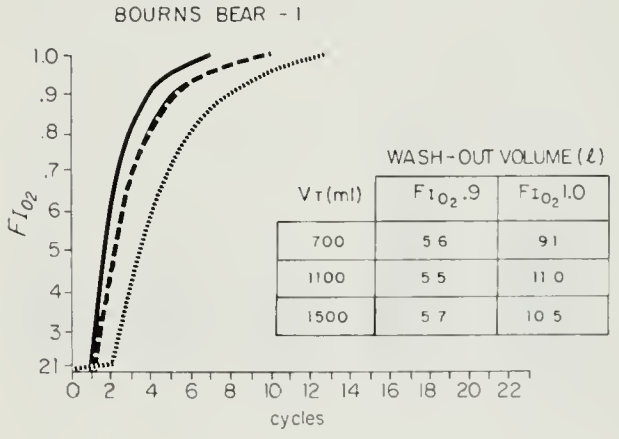

Fig. 6. Wash-out curves and calculated wash-out volumes to $F_{I_{O_2}}$ 0.9 and 1.0 for the Bourns BEAR-1, using V_T 700 ml (solid line), 1,100 ml (heavy dashed line), and 1,500 ml (fine dashed line).

the ventilator was set at an indicated 100 per cent oxygen, a measured $F_{I_{O_2}}$ of 0.90 was attained at 1.3 l lower wash-out volume (8.4 vs 9.7 l) than with room air; wash-out time to $F_{I_{O_2}}$ 0.90 would be approximately 1 minute in both cases.

Wash-out curves at tidal volumes 700, 1,100, and 1,500 ml for each of the ventilators tested are shown in Figures 3-7, with room air used as the gas washed out. The volume of 100 per cent oxygen required to wash out the ventilator and tubing tended to increase with increasing tidal volume and ranged from 6.3 l for the Monaghan 225 at tidal volume 700 ml, to 18.0 l for both the Bennett MA-1 and the Ohio 560 at tidal volume 1,500 ml. To $F_{I_{O_2}}$ 0.90 the volumes varied from 2.8 l for the Monaghan 225 at tidal volume 700 ml to 10.5 l for the Ohio 560 at tidal volume 1,500 ml.

As can be seen from Figures 3-7, the smallest tidal volume was associated with the smallest wash-out volume but with the greatest number of

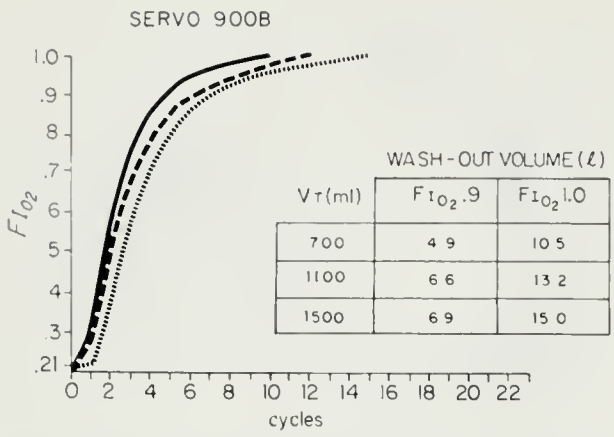

Fig. 7. Wash-out curves and calculated wash-out volumes to FI_{O_2} 0.9 and 1.0 for the Servo 900-B, using VT 700 ml (solid line), 1,100 ml (heavy dashed line), and 1,500 ml (fine dashed line).

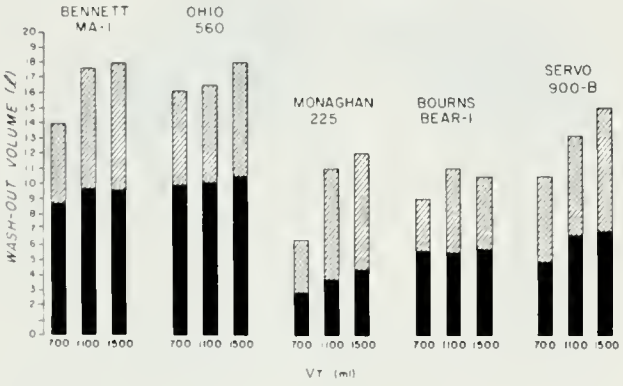

Fig. 8. Comparison of wash-out volumes to FI_{O_2} 0.9 (shaded area) and 1.0 (hatched area) at three tidal volumes for each ventilator studied.

cycles, so that if rate were held constant, wash-out to the desired FI_{O_2} would take considerably longer with smaller tidal volumes.

For comparison, wash-out volumes to FI_{O_2} 0.90 and 1.00 for each ventilator studied are shown graphically for each tidal volume used in Figure 8.

Discussion

The considerable variation in wash-out volume observed among five different models of ventilators does not appear to be explained by differences in the mechanism of delivery. For example, the Bennett MA-1, Ohio 560, and Monaghan 225 ventilators utilize a canister and bellows, while the Bourns BEAR-1 and Servo 900-B do not. The Monaghan 225, which has the largest canister, required the smallest wash-out volume. No attempt was made to estimate total circuit volume, although differences in this and in tubing volume may account for the differences observed among ventilator models.

While a theoretical consideration of time-constants and other factors influencing gas concentrations in ventilator circuits is beyond the intended scope of this descriptive study, our observations have practical clinical implications. They demonstrate that, for the five ventilators tested, a significant volume of gas must be washed out before a dialed FI_{O_2} of 1.0 can be delivered to the patient. This washout may take one or two minutes, depending upon tidal volume and cycling frequency. If the ventilator is used for preoxygenation, endotracheal suction-related hypoxemia may not be prevented if suctioning is initiated before wash-out of gas is complete.

Acknowledgment

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Air Entrainment Masks: Jet Mixing Is How They Work; The Bernoulli and Venturi Principles Are How They Don't

Robert Scacci PhD

Three primary misconceptions about the air entrainment process used in some oxygen therapy masks are that (1) the Bernoulli principle causes air entrainment, (2) air is entrained by a pressure difference induced by the primary (jet) flow, (3) increasing the primary flow of oxygen increases the delivered oxygen concentration. The principle of air entrainment is constant-pressure jet mixing. The Bernoulli principle is not the basis for this mixing, because it states that pressure is reduced in exchange for an increase in velocity. Entrainment occurs at constant pressure; there is no mechanism to support a pressure gradient between the jet and the entrained air. Air entrainment occurs as a consequence of fluid viscosity. The amount of entrained air is proportional to the primary (jet) flow, leaving the concentration of oxygen unaffected by changes in primary flow.

Oxygen administration with air entrainment masks has become more routine with increasing availability of these items. These masks were originally called venturi masks because they were said to use the Venturi principle. Many therapists and physicians now know that these masks do not work by the Venturi principle, but by air entrainment.

The purpose of this paper is to dispel the following three misconceptions about the entrainment process in air entrainment masks:

1. The Bernoulli principle causes air entrainment.
2. Air is entrained by a pressure difference induced by the primary (jet) flow.
3. Increasing the primary flow of oxygen increases the delivered oxygen concentration.

The principle of air entrainment is constant-pressure jet mixing. Mixing of a moving stream (the oxygen jet flow) with a quiescent fluid (room air) occurs as a consequence of fluid *viscosity*, not a pressure gradient. This will be explained in detail later.

Before explaining constant pressure jet mixing, I will discuss the Bernoulli and Venturi principles, because an understanding of these concepts will clarify why air entrainment masks are *not* venturi masks and *do not* work on the Bernoulli principle.

The Bernoulli Principle

In 1730 Daniel Bernoulli recognized that steady, incompressible, frictionless, fluid flow obeyed the first law of thermodynamics (*ie*, the law of conservation of energy), and he stated the first law in terms of fluid mechanics. For isoenergetic flow (*ie*, no heat or energy added to or taken from the flow), and in the absence of gravitational effects (no large elevation changes), Bernoulli's principle simply states that along any fluid streamline, pressure energy and velocity energy are related according to the following equation:

$$P_1 - P_2 = \tfrac{1}{2} \rho (V_2^2 - V_1^2)$$

where the subscripts 1 and 2 are two points along a streamline, ρ is fluid density, and V is velocity. This

Mr Scacci is Technical Director, Pulmonary Laboratories, Methodist Hospitals of Dallas, Dallas, Texas.

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Reprint requests to: Robert Scacci PhD, Methodist Hospitals of Dallas, PO Box 225999, Dallas TX 75265.

means that pressure is reduced in exchange for an increase in velocity, and vice versa. Hence, the Bernoulli principle simply describes the pressure/velocity trade-off necessary to conserve total fluid energy.

The Venturi Tube

In 1767 Giovanni Venturi described a tube intended to measure fluid flow. He knew that one could calculate the velocity of the fluid from Bernoulli's equation by measuring the pressure at two points. To induce a pressure change, he constricted a tube (Fig. 1) and made two pressure taps—one upstream, represented by point 1 in Figure 1, and one at the throat, represented by point 2. The tube constriction forces the fluid to accelerate to a higher velocity to squeeze through the throat. The pressure at the throat decreases according to the Bernoulli equation. Venturi could readily measure the pressure differential, from which he could calculate the velocity. Some unavoidable pressure loss in the tube occurs because of friction.

Thus the venturi tube is basically a flowmeter, but it is not used in most hospital applications, because velocity varies with the square root of pressure.

The venturi tube may be used as a fluid entrainment device, *although it is not used for oxygen therapy masks*. An example of a venturi entrainment device is the carburetor in a car engine, schematically depicted in Figure 2. The upstream end of the tube (mouth of the carburetor) is at ambient pressure; consequently, because of Bernoulli's principle, the pressure at the throat will be less than ambient when there is flow through the tube. This pressure gradient between the fuel chamber and the throat causes fuel to be lifted to the level of the throat and sucked into the air flow. Obviously this is not the geometric configuration of an air entrainment mask. Figure 3 is the basic geometry of a jet air entrainment device, which consists of the primary jet-flow nozzle, air inlet ports, and a mixing chamber. This shows an entirely different fluid entrainment mechanism from the venturi tube described.

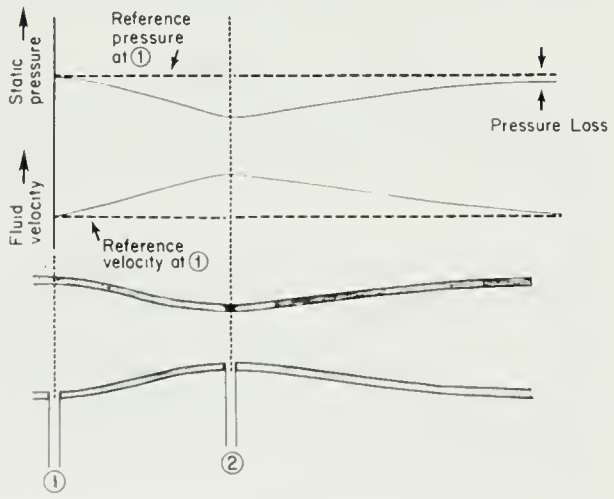

Fig. 1. Venturi tube showing axial variation of velocity and pressure. Except for slight pressure loss due to friction, the pressure and velocity at any two points follow Bernoulli's principle: $P_1 - P_2 = \frac{1}{2}\rho (V_2^2 - V_1^2)$

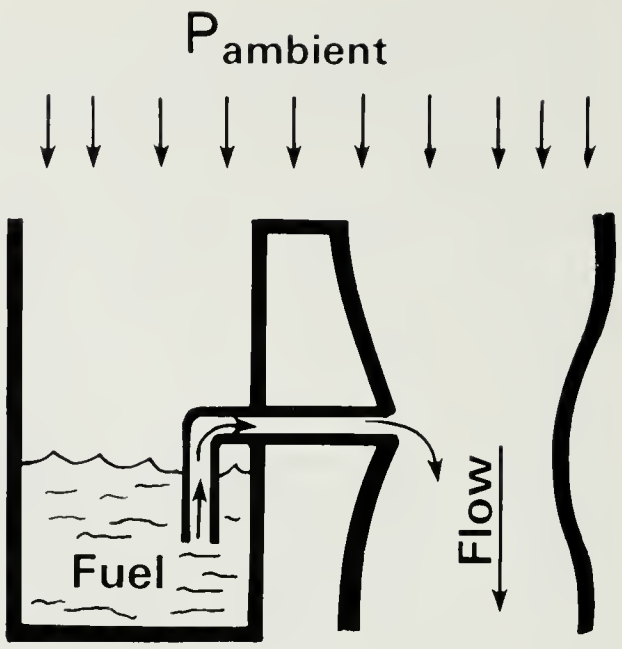

Fig. 2. The automotive carburetor, a venturi mixing device, operates by Bernoulli's principle. Entrained fluid (fuel) enters main flow because of pressure gradient induced by throat of venturi tube. Note that geometry of tube and entrainment orifice is not similar to that of oxygen therapy masks.

The Jet Mixer

In Figure 3 the jet nozzle is the converging half of a venturi tube. Fluid in the nozzle obeys Bernoulli's principle, as does fluid in the converging half of the

venturi tube. Unlike a venturi tube, the nozzle exit is in or very close to the plane of the entrainment port, and this causes the pressure at the nozzle exit to be essentially ambient, regardless of the jet flow. The nozzle exit, the entrainment ports, and the mixing barrel are always at ambient pressure, if one neglects the very slight pressure needed to move flow through the mixing barrel. Varying the jet flow changes the pressure *only in the nozzle*, as shown in the upper part of Figure 3. Entrainment occurs at constant pressure because there is simply *no mechanism available to support a pressure gradient* between the jet and the entrained air.

Oxygen therapy masks look different from Figure 3 but are basically the same, consisting of the primary jet-flow nozzle, air inlet ports, and mixing chamber. In therapy masks the entrainment ports restrict the entrance of ambient air to varying degrees, because restricting the entrainment port is the mechanism by which higher oxygen concentrations are obtained. These conditions may cause the nozzle exit pressure to be slightly less than the pressure in the entrainment port. However, any such pressure gradient is *insignificant to the entrainment process*, and air entrainment occurs at essentially *constant pressure*.

At this point, we may ask, "If pressure differences do not force room air into the entrainment ports, what does?" Let us consider an analogy.

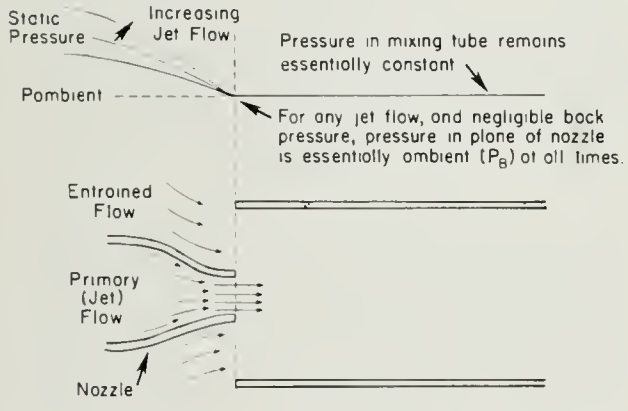
Static Pressure
Increasing Jet Flow
Pressure in mixing tube remains essentially constant
Pambient
For any jet flow, and negligible back pressure, pressure in plane of nozzle is essentially ambient (P_B) at all times.
Entrained Flow
Primary (Jet) Flow
Nozzle

Fig. 3. Jet mixing device, showing axial variation of pressure. In the nozzle, pressure follows Bernoulli's principle, but nozzle exit, entrainment ports, and mixing tube are always at ambient pressure. Entrained fluid is drawn into the mixing tube by viscous interaction with primary (jet) flow. Note that oxygen therapy masks contain the same basic elements of nozzle, entrainment ports, and mixing chamber.

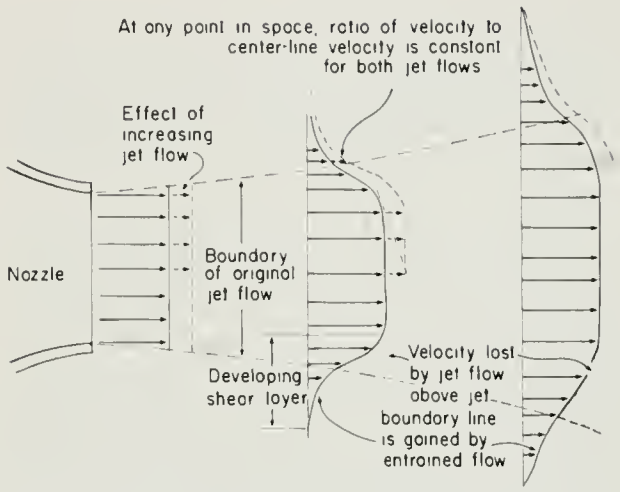
At any point in space, ratio of velocity to center-line velocity is constant for both jet flows
Effect of increasing jet flow
Nozzle
Boundary of original jet flow
Developing sheor layer
Velocity lost by jet flow above jet boundary line is gained by entrained flow

Fig. 4. Development of velocity profiles in jet mixing. Fluid velocity at three planes in space is indicated by length of arrows. Viscosity causes original jet to lose velocity while quiescent fluid gains velocity. Shear layer widens as more of original jet velocity is imparted to more entrained fluid. Broken lines indicate effect of increasing jet flow. At any point in space, the ratio of the velocity at that point to the center-line velocity is constant for either flow. Consequently, the ratio of entrained mass flow to jet mass flow is constant also. If the jet is oxygen and the entrained flow is air, then downstream oxygen concentration is independent of jet flow, as in oxygen therapy masks.

Imagine someone falling out of a moving vehicle. Upon his hitting the road surface, friction between the victim and the road will cause the road to successfully decelerate the victim. The force of the victim's moving body will unsuccessfully try to accelerate the road. The moving/nonmoving interface between the victim and road cannot be sustained without something happening, and the result is shearing of clothing and almost certainly some of the hide from the hapless victim.

Now, if a moving stream of fluid encounters a stationary or quiescent fluid, similar effects occur. Viscosity will account for much of the friction that causes the quiescent fluid to decelerate the moving fluid, and that causes the moving fluid to accelerate the quiescent fluid. The shearing is not as violent as the analogy states, because fluid more readily deforms than do people or roads.

The viscous shearing between the moving and static fluid layers causes an exchange of velocity; the moving fluid gives up some velocity while the

nonmoving mass acquires some velocity (Fig. 4). In essence, the entrained air is dragged into the moving stream. This process occurs at constant pressure. Very small pressure gradients occur within the quiescent fluid, causing more fluid to move to the interface.

In an entrainment mask, partial occlusion of the entrainment ports may create a slightly negative pressure inside the mask. This is a result of the jet's attempting to drag away more air per unit of time than can be supplied through the occluded port. The pressure gradient in this case is a *consequence* of constant pressure jet mixing and not the *cause* of such mixing. At this point it should be clear that Bernoulli's law relating pressure and velocity and Venturi's flowmeter tube are not related to the jet mixing process.

Several investigators[1-3] have shown experimentally that delivered oxygen concentration is relatively constant for wide ranges of primary flow.

It is relatively easy for fluid dynamicists to predict the amount of mass entrainment for any jet flow. Obviously, the calculations for jet mixing are beyond the scope of this paper, but they can be found in a doctoral thesis* that is a condensed version of the works of Korst, Page, and Childs,[4] Hill,** and Nash.[5]. These calculations are for turbulent jet mixing and are appropriate because turbulent flow usually occurs in air entrainment masks.

Theoretical calculations show that the ratio of the velocity at any point in space, to the centerline velocity of the jet, is a constant for any value of the jet velocity. Hence, the ratio of mass entrained to the primary jet mass flow must also be a constant. Thus, the delivered oxygen concentration, which is a function of the mass entrainment ratio, is expected to be constant in the presence of various flow values. These results are appropriate to the flow models used by theorists. Results with an actual entrainment mask may depart from theory, especially with inordinately high or low oxygen flows. Users should, of course, follow manufacturer's guidelines governing the flow setting.

Two conditions may cause delivered oxygen concentration to deviate from rated values. First, should the downstream pressure in the tubing-mask system increase for any reason, this increased pressure may propagate upstream as far as the entrainment ports. If the pressure at the entrainment ports exceeds ambient pressure, no air can enter. Hence, with sufficient back pressure, the delivered oxygen concentration will rapidly approach 100%. Second, respiratory therapy personnel must make certain that the total flow going to the patient exceeds his peak inspiratory flow demand. Otherwise, the patient will draw more room air from the entrainment ports than is entrained by the jet flow, lowering oxygen concentration.

Summary

The Bernoulli principle describes the pressure/velocity relationship that exists in certain fluid flows. This relationship is not responsible for air entrainment in an oxygen therapy mask, nor is the venturi tube in any way related to such entrainment. Air entrainment occurs as a consequence of fluid viscosity. The viscous shearing force that exists between moving and static layers of fluid causes the non-moving fluid to be dragged into the moving stream. Any pressure gradients that may exist are secondary to the mixing process. Experimental results prove the amount of entrained air to be proportional to the primary (jet) flow. Consequently, the concentration of the delivered gas is relatively unchanged when the primary flow is changed.

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Subjective Effects of Dry versus Humidified Low Flow Oxygen

William Estey RN

Of 34 hospitalized patients receiving low flow oxygen (1 to 4 l/min) continuously by nasal cannula, 19 randomly selected patients breathed humidified oxygen and 15 randomly selected patients breathed dry oxygen. During a 5- to 10-day study period the patients answered questions about subjective findings of dry nasal passages, dry throat, headache, chest discomfort, cold symptoms, and changes in sputum production. No significant difference was found in any of these complications between the patient group breathing humidified oxygen and the group breathing dry oxygen. Because of the apparent lack of problems attributable to inhalation of dry low flow oxygen, and because of the cost of humidifying low flow nasal oxygen, our hospital has discontinued humidifying oxygen administered in this way.

Introduction

Current medical practice suggests that medical oxygen should be humidified prior to patient use.[1] A normally functioning nasopharynx should be able to raise the relative humidity of ambient air to 75%; however, it has been assumed that this would not occur during administration of low flow nasal oxygen.[2] Data are scarce on the effects of humidifying oxygen administered by nasal cannula at low flows; most studies have dealt with patients whose upper airways were bypassed by endotracheal[3] or tracheostomy[4] tubes.

Clinical experience indicates that certain side-effects may be associated with the inhalation of low flow nasal oxygen. These side-effects include dry nose or throat, dryness or burning of the eyes, headache, chest discomfort, and infections of the nose, throat, and sinuses. We have been impressed that no objective evidence has shown that humidification of low flow oxygen alters the incidence of these side-effects. In view of the high costs of humidifying oxygen at the hospital bedside, we wished to determine whether such humidification was really necessary. We therefore conducted a study to determine whether patients derived subjective benefit from the addition of humidity to oxygen delivered continuously by nasal cannula.

Methods and Materials

We studied 34 patients who were receiving oxygen continuously by nasal cannula at up to 4 liters per

Mr Estey is Pulmonary Nurse Clinician, Pulmonary Rehabilitation Program, Bishop Clarkson Memorial Hospital, Omaha, Nebraska.

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minute. The patients were randomly assigned to two groups: 19 patients received humidified oxygen; 15 received dry oxygen. Oxygen was administered via Hudson No. 1104 cannulas (Hudson Oxygen Therapy Sales Co, Temecula CA). Humidification, when supplied, was accomplished with Aquapak No. 411 prefilled humidifiers and No. 011 adapters (Respiratory Care Inc, Arlington Heights IL).

For a 5- to 10-day period all patients were examined daily and asked a battery of standard questions each day; the questions, answered by Yes or No, elicited information on consecutive days about symptoms and side-effects. These, in the order of questioning, included the presence of (1) dry nasal passages, (2) dry throat, (3) headache, (4) chest discomfort, (5) common cold symptoms, and (6) increase or change in sputum production. Each question was treated as a separate variable and was analyzed by a separate analysis of variance with repeated measures (treatments by days).

Results

The results indicated no significant difference in numbers of any category of complications between the patient groups receiving humidified and dry oxygen (Table 1). No significant main effect for days was found in any of the six separate analyses.

Table 1. Numbers of Various Complications over 5-Day Period in Humidified Oxygen and Dry Oxygen Patient Groups

Complications	Mean Numbers of Complications*	
	Humidified O_2 Group (n = 19)	Dry O_2 Group (n = 15)
Dry Nose	2.73	2.07
Dry Throat	2.84	1.40
Headache	0.26	0 20
Chest Discomfort	0.68	0.13
Common Cold Symptoms	1.05	0.27
Sputum Production Change	1.05	0 33

*None of the differences between means is statistically significant

Discussion

We embarked on this study to determine whether patients derived significant benefit from humidification of low flow oxygen. Our results indicated that no advantages are apparent in using humidity in this form of therapy. The group of patients using humidified oxygen had more complications related to the questionnaire than did the group using dry oxygen; however, the difference was not statistically significant. Also, no pattern of complications over days was found.

A secondary purpose of this study was to determine whether the humidification of low flow oxygen is cost-justified. We have no available data on the national cost of humidifying low flow oxygen, but at our 550-bed hospital the annual cost approaches $12,000 for supplies alone.

The implications of this study's results are that without low flow oxygen humidification, the financial outlay for prefilled disposable humidifiers could be minimized, reducing health care costs. Based on these results, we do not plan to continue humidifying low flow oxygen at our hospital.

These results force us to review current practices and further evaluate humidification in oxygen therapy. Because this is the only study of its kind, of hospitalized patients or others, that is known to us, we plan to study the same topic in outpatients receiving continuous low flow oxygen by nasal cannula over a long period of time. This should permit us to work with a larger sample, longer periods of oxygen use, and an expanded questionnaire.

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Use of the Concept of Ventilator Compliance in the Determination of Static Total Compliance

Robert R Demers BS RRT, Melvin R Pratter MD, and Richard S Irwin MD

Static total compliance (Cst) provides a noninvasive means of determining the mechanical properties of the respiratory system at the bedside. Together with certain other indices of cardiorespiratory function, Cst is often useful in determining the best level of positive end-expiratory pressure (so-called "optimum PEEP") to employ in the treatment of a particular patient. The accurate and precise measurement of Cst, in turn, requires the correct technique for measuring tidal volume and the simultaneous change in the static pressure that accompanied the delivery of that volume. Consequently, for patients being mechanically ventilated, the ventilator's compressible volume must be determined and substracted from the total volume exhaled through a spirometer during a single breathing cycle. The change in pressure, measured statically, that is divided into the tidal volume figure is defined as the "plateau pressure" minus the end-expiratory pressure. This presentation consists of a discussion of physiologic concepts, practical hints, and actual clinical examples relating to the measurement of Cst. (Demers RR, Pratter MR, Irwin RS. Use of the Concept of Ventilator Compliance in the Determination of Static Total Compliance. Respir Care 1981;26:644-648. Key words: Critical care, Data collection, Lung compliance, Positive pressure respiration, Respiration, artificial, Respiratory function tests, Respiratory therapy.)

Introduction

Various studies have demonstrated that the elastic recoil of the lung and chest wall measured under static conditions is a very useful value to monitor.[1,2] This index is termed "static total compliance" because the measurement is performed under *static*, as opposed to dynamic, conditions and because the recoil pressure that one measures is attributable to the total respiratory system (both the chest wall and the lungs). *Compliance* is the term for the measure of how readily the pulmonary system complies with or yields to a distending pressure. During therapy with positive end-expiratory pressure (PEEP), static total compliance data are especially useful because of their value in determining the best PEEP level, so-called "optimum PEEP," to employ for a particular patient.[3] Because we have observed that static total compliance is frequently miscalculated and misunderstood by respiratory therapists and physicians, we decided to review the topic.

Mr Demers is Pulmonary Staff Assistant and Instructor in Medicine; Dr Pratter is Director, Pulmonary Function Laboratory, and Assistant Professor of Medicine; and Dr Irwin is Director, Division of Pulmonary Medicine, and Associate Professor of Medicine—University of Massachusetts Medical Center and Medical School, Worcester, Massachusetts.

Reprint requests: Robert R Demers RRT, University of Massachusetts Medical Center, Worcester MA 01605.

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Concepts

Calculation of Tidal Volume

The term "tidal volume" was coined by pulmonary physiologists some years ago because they con-

sidered spontaneous breathing to be reminiscent of the ebb and flow of ocean tides. "Tidal volume" and "tidal ventilation" are terms for the volume of gas exhaled during a single breathing cycle. Not uncommonly, some assume that the volume of gas inhaled is identical to the volume of gas exhaled. This is not strictly correct. Inhaled gases that are cooler than 37°C undergo thermal expansion as predicted by Charles' Law. Furthermore, inhaled gases that are less than completely saturated with water vapor tend to increase in volume with the addition of more water vapor. Even if the inhaled gases were completely saturated with water vapor at a temperature of 37°C, a volumetric difference between inhaled and exhaled gases would still persist. This difference between inhaled and exhaled volumes is attributable to the fact that the volume of oxygen taken up from the lung in the process of gas exchange exceeds the volume of carbon dioxide excreted by the lung. For example, in a normal subject at rest, oxygen consumption is about 250 ml/min, while carbon dioxide production is about 200 ml/min. Therefore, in patients who are breathing spontaneously, the precise volume expelled per ventilatory cycle is, indeed, definable as tidal volume. On the other hand, tidal volume is *not* quantitatively identical to the volume of gas expelled from the ventilator exhalation manifold in patients who are being mechanically ventilated with positive-pressure machines. Mechanical ventilators and their internal housings, humidifiers, and tubing systems incorporate some space that contains gases, and these gases obey Boyle's Law.[4] Thus, as pressure increases during positive-pressure inflation, some of the gases expelled from the bellows, piston, and valve box are sequestered in the tubing system and do not actually reach the patient. If gases are measured volumetrically as the machine cycles into the expiratory phase, the volume of gas collected per cycle actually represents a combination of this so-called "compressible volume" of the machine and the true tidal volume of the patient.

Appropriate calculations can be employed to compute the compressible volume and compensate for it in order to generate a correct tidal volume figure. This calculation requires the use of a number, the "ventilator compliance," that applies to a particular brand and model of ventilator fitted with a specific type of tubing and humidifier.[5] For example, in the following case an MA-1 ventilator (Puritan-Bennett Corp, Kansas City MO) is being used:

Example 1

A respiratory therapist is performing a routine check to assure that a ventilator is functioning properly before employing it for a surgical patient. A determination of ventilator compliance, described as follows, is part of the routine checking procedure. A Breathometer (JH Emerson Co, Cambridge MA), an accurate spirometer,[6] is connected to the ventilator's exhalation manifold. Next, the therapist completely occludes the patient connection port (that part of the "patient Y" that is normally fitted to the endotracheal or tracheostomy tube) and manually cycles the ventilator. During 10 ventilatory cycles, 2,100 ml are measured at the spirometer; end-inspiratory pressure is observed to be 70 cm H_2O, and end-expiratory pressure is observed to be 0 cm H_2O. Thus, the volume expelled from the system per cycle is:

$$\text{Spirometer volume} = 2{,}100 \text{ ml}/10 \text{ breaths} = 210 \text{ ml/breath}$$

This entire volume represents the compressible volume because the tidal volume has been rendered zero by occlusion of the patient connection port. Ventilator compliance (Cv) is defined as the ratio between the observed compressible volume (Vc) and the observed pressure change (ΔP) that occurs simultaneously:

$$Cv = \frac{Vc}{\Delta P}$$

In this particular case,

$$Cv = \frac{210 \text{ ml}}{70 \text{ cm } H_2O}$$

$$= 3.0 \text{ ml/cm } H_2O$$

This figure indicates that for every cm H_2O pressure applied to the system between end-expiration and end-inspiration, 3 ml of gas will be sequestered in the ventilator's tubing system. The empirical determination of ventilator compliance is quickly and easily performed at the bedside and provides the

number that can subsequently be used to calculate true tidal volumes.

It should be noted here that the effects of resistance under the conditions described in this example are negligible. Because no bulk flow from the system occurs, the pressure observed and recorded is entirely attributable to the elastic recoil of the ventilator tubing system and the gases incorporated therein. In other words, "zero flow" or "static" conditions prevail in such circumstances.

Clinical application of this concept is illustrated in the following example.

Example 2

A patient is being mechanically ventilated with the Bennett MA-1 for treatment of the adult respiratory distress syndrome. End-inspiratory pressure (which coincides with peak pressure during normal cycling of the MA-1) is observed to be 50 cm H_2O, end-expiratory pressure is 0 cm H_2O, and a volume of 500 ml per cycle is being expelled from the exhalation manifold. Rearrangement of the previous equation yields the following:

$$Vc = Cv \times \Delta P$$

In this particular case,

$$\begin{aligned} Vc &= (3 \text{ ml/cm } H_2O) \times (50 - 0 \text{ cm } H_2O) \\ &= 150 \text{ ml} \end{aligned}$$

The patient's actual tidal volume is the difference between the spirometer volume and the compressible volume as calculated earlier:

TV = spirometer volume − compressible volume

Again, in this particular case,

$$\begin{aligned} TV &= 500 \text{ ml} - 150 \text{ ml} \\ &= 350 \text{ ml} \end{aligned}$$

Therefore, the therapist would chart that the patient's tidal volume was 350 ml, and *not* 500 ml, and would perhaps make an entry to indicate that a correction had been performed.

Calculation of Static Total Compliance

If a calculation of the patient's static total compliance were requested at this time, an easy way of producing a static or zero-flow condition would be to cap the expiratory limb of the ventilator manually after the delivery of a breath. If this is done, one observes that the needle of the pressure gauge peaks at the same level as before, but then momentarily stabilizes ("plateaus") after inspiratory flow ceases. Consider the same patient for whom a static total compliance procedure is now performed:

Example 3

Peak pressure (which now does *not* coincide with end-inspiration) is observed still to be 50 cm H_2O, plateau pressure is observed to be 20 cm H_2O, end-expiratory pressure is 0 cm H_2O, and a volume of 500 ml per cycle is measured at the exhalation manifold. Compressible volume is now:

$$\begin{aligned} Vc &= (3 \text{ ml/cm } H_2O) \times (20 - 0 \text{ cm } H_2O) \\ &= 60 \text{ ml} \end{aligned}$$

Tidal volume is now calculated in the same fashion as outlined earlier:

$$\begin{aligned} TV &= 500 \text{ ml} - 60 \text{ ml} \\ &= 440 \text{ ml} \end{aligned}$$

Notice that the imposition of an inspiratory hold or pause has resulted in a slight increase in tidal volume. Static total compliance is now calculated as follows:

$$Cst = \frac{TV}{\Delta P}$$

where TV is the corrected tidal volume and ΔP is the observed change in pressure between end-inspiration and end-expiration. In this particular case,

$$Cst = \frac{440 \text{ ml}}{20 - 0 \text{ cm } H_2O}$$

$$= 22 \text{ ml/cm } H_2O$$

During therapy with positive end-expiratory pressure (PEEP), the calculation of corrected tidal volumes and static total compliance figures is subject to further modification. If the use of PEEP or increase in PEEP does succeed in recruiting alveoli that were previously nonventilated or underventilated, the measured compliance can be expected to increase. If, on the other hand, excessive amounts of PEEP are used, the lung and chest wall will become

"stiffer" and measured compliance will decrease. Example 4 is the same patient under the following conditions:

Example 4

A PEEP level of 5 cm H_2O is added to the patient previously discussed. Peak pressure is observed to be 55 cm H_2O, plateau pressure is observed to be 23 cm H_2O, and 500 ml per cycle continues to be measured at the exhalation manifold. Compressible volume is:

$$Vc = (3 \text{ ml/cm } H_2O) \times (23 - 5 \text{ cm } H_2O)$$
$$= 54 \text{ ml}$$

Tidal volume is now:

$$TV = 500 \text{ ml} - 54 \text{ ml}$$
$$= 446 \text{ ml}$$

Static total compliance is computed as before:

$$Cst = \frac{446 \text{ ml}}{(23 - 5) \text{ cm } H_2O}$$

$$= 24.8 \text{ ml/cm } H_2O$$

Notice that the patient's static total compliance has marginally increased, indicating that the imposition of PEEP has succeeded in recruiting more alveoli, a favorable response.

Example 5

The attending physician decides to increase the PEEP level to 10 cm H_2O and requests that static total compliance be recalculated at this PEEP level so he can attempt to assess the patient's response. At the 10 cm H_2O PEEP level, peak pressure is observed to be 60 cm H_2O, plateau pressure is measured and found to be 25 cm H_2O, and the spirometer volume remains constant at 500 ml per cycle. Compressible volume is calculated:

$$Vc = (3 \text{ ml/cm } H_2O) \times (25 - 10 \text{ cm } H_2O)$$
$$= 45 \text{ ml}$$

Tidal volume is:

$$TV = 500 \text{ ml} - 45 \text{ ml}$$
$$= 455 \text{ ml}$$

Static total compliance has increased further to:

$$Cst = \frac{455 \text{ ml}}{(25 - 10) \text{ cm } H_2O}$$

$$= 30.3 \text{ ml/cm } H_2O$$

Example 6

The patient's favorable response to increasing PEEP prompts the attending physician to order that PEEP be increased further to 15 cm H_2O. At this PEEP level, peak pressure is 63 cm H_2O, plateau pressure is 36 cm H_2O, and spirometer volume remains at 500 ml per cycle. Compressible volume is:

$$Vc = (3 \text{ ml/cm } H_2O) \times (36 - 15 \text{ cm } H_2O)$$
$$= 63 \text{ ml}$$

Tidal volume is:

$$TV = 500 \text{ ml} - 63 \text{ ml}$$
$$= 437 \text{ ml}$$

Finally, static total compliance is now observed to have fallen:

$$Cst = \frac{437 \text{ ml}}{(36 - 15) \text{ cm } H_2O}$$

$$= 20.8 \text{ ml/cm } H_2O$$

This decrease in measured compliance as compared to the value calculated at the 10 cm H_2O level would prompt the physician to reinstitute the level of PEEP (10 cm H_2O) that resulted in the highest measured static total compliance figure.

Notice that peak pressure is irrelevant in the calculation of static total compliance because peak pressure does not prevail at end-inspiration during the performance of the procedure by which Cst is measured.[7] However, while the patient is being ventilated in the usual fashion and in the absence of an inspiratory pause, tidal volume and compressible volume will change accordingly. For example, in the situation described in Example 6, peak pressure was 63 cm H_2O, PEEP was 15 cm H_2O, and spirometer volume was 500 ml. Compressible volume would be:

$$Vc = (3 \text{ ml/cm } H_2O) \times (63 - 15 \text{ cm } H_2O)$$
$$= 144 \text{ ml}$$

A corresponding change in tidal volume will be observed:

$$TV = 500 \text{ ml} - 144 \text{ ml}$$
$$= 356 \text{ ml}$$

This figure differs appreciably from the tidal volume observed during the compliance measurement procedure, and a tidal volume number of 356 ml would be charted during routine checking of the patient in the absence of an inspiratory hold.

Discussion

Some controversy still persists regarding the role of static total compliance as a monitoring tool during PEEP. Although Suter *et al*[2] demonstrated excellent correlation of the static total compliance figure with other, more invasive, indices of cardiorespiratory function, one cannot anticipate that static total compliance will reveal the optimum PEEP level under all conditions.[3] Therefore, we recommend that practitioners not only pay heed to serial measurements of static total compliance, but also perform periodic determinations of mixed venous oxygen tension and cardiac output.

Needless to say, if one makes the effort to measure static total compliance in the first place, one should perform the calculation appropriately. This implies that the true tidal volume should be determined (spirometer volume minus compressible volume), and the *change* in pressure (plateau pressure minus end-expiratory pressure) that is observed during tidal ventilation should be recorded. The magnitudes of error one could incur by making certain common errors are demonstrated in Table 1. In the table, a "Type I Error" represents the compliance figure one would obtain if spirometer volume (compressible volume plus tidal volume) were divided by peak pressure in four of the six examples cited in the text. A "Type II Error" is the error that would obtain if one attempted to calculate compliance as the ratio between tidal volume (calculated correctly) and plateau pressure (assuming one neglected to subtract end-expiratory pressure) in each of the examples. The systematic error introduced by committing either of these types of mistakes is strikingly evident.

Table 1. Static Total Compliance (Cst) as Determined by Various Methods

Example Number	Actual Cst (ml/cm H_2O)	Cst with Type I Error (ml/cm H_2O)	Cst with Type II Error (ml/cm H_2O)
3	22.0 $(\frac{440}{20})$	10.0 $(\frac{500}{50})$	22.0 $(\frac{440}{20})$
4	24.8 $(\frac{446}{18})$	9.1 $(\frac{500}{55})$	19.4 $(\frac{446}{23})$
5	30.3 $(\frac{455}{15})$	8.3 $(\frac{500}{60})$	18.2 $(\frac{455}{25})$
6	20.8 $(\frac{437}{21})$	7.9 $(\frac{500}{63})$	12.1 $(\frac{437}{36})$

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Sound Levels inside Incubators and Oxygen Hoods Used with Nebulizers and Humidifiers

Richard W Beckham MEd RRT and Shelley Cominsky Mishoe MEd RRT

Degree of hearing loss in the infant is related to high-intensity or high-frequency sound (noise). We measured sound levels of 6 models of pneumatic nebulizers and 8 models of humidifiers at various flowrates, F_{IO_2} settings, and water levels, when they were attached to incubators and oxygen hoods; we also evaluated sound levels from 2 models of ultrasonic nebulizers at various output settings. Among the pneumatic nebulizers, the recommended 58-dbA maximum sound level was exceeded by the Aquapak 621, Ohio Deluxe, and Puritan All-Purpose devices. The Bard Parker, Foregger, and Inspiron pneumatic nebulizers' sound levels were 58 dbA or below except during the dry water-level condition. Among the humidifiers, all produced sound levels below the recommended 58-dbA maximum except the Travenol humidifier; the Bennett Cascade, Conchapak, Foregger, and Hudson humidifiers produced mean sound levels in the 43.0- to 43.5-dbA range. Sound levels increased under all conditions when the devices were used with oxygen hoods. The ultrasonic nebulizers produced low sound levels, but the high-frequency ultrasound they also produce may be undesirable for infants; this question requires further investigation. (Beckham RW, Mishoe SC. Sound Levels inside Incubators and Oxygen Hoods Used with Nebulizers and Humidifiers. Respir Care 1982;27:33-40. Key words: Acoustic stimulation, Critical care, Environment, controlled, Environmental health, Environmental pollution, Equipment and supplies, hospital, Hearing loss, noise-induced, Incubators, infant, Infant care, Neonatology, Noise, Preventive medicine, Respiratory therapy, Sound.)

Introduction

According to the Environmental Protection Agency, millions of persons in the United States suffer from some degree of hearing loss directly caused by noise.[1] However, hearing loss is not the only noise-related hazard. Excessive noise can also cause heart-rhythm changes, dilation of blood vessels, and increased blood pressure.[1,2] In addition, the ability of the eyes to focus may be impaired, certain glands may increase hormone production, and the stomach may secrete more acid.[1]

Experimental and clinical evidence has indicated that the developing hearing organ of the infant has a greater susceptibility to noise than do the hearing organs of older children and adults.[3-6] This relationship is thought to result from the stage of development of the infant's immature organ of corti, but evidence also indicates that even when older persons are exposed to the same sound levels, there probably exists a relationship between age and degree of trauma.[3,7] In one study of two groups of industrial workers, respectively aged 18-29 and 30-

Mr Beckham is Chairman/Associate Professor, and Ms Mishoe is Clinical Coordinator/Assistant Professor, Department of Respiratory Therapy, Medical College of Georgia, Augusta, Georgia.

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45 years, the noise-induced hearing loss was three times greater in the younger group than in the older group after 3 years' exposure of both groups to the same noise levels.[7]

Experimental and clinical evidence also has demonstrated the hazardous effects on the cochlea of noise in combination with ototoxic antibiotics. Light microscopy shows that the damage caused by the combination of noise and ototoxic antibiotics is characterized by degeneration of the sensory cells of the organ of corti.[8-10] The potentiation of noise-induced hearing loss by ototoxic aminoglycosidic antibiotics has been established in animal studies, and it has been found that the cellular damage to the organ of corti after combined exposure to noise and the antibiotics exceeds that of the sum of the separate effects of the agents.[2,9] In experimental animals, levels as low as 58 decibels as measured on the A-weighted scale (dbA), in themselves benign, potentiated the risk of hearing loss from ototoxic drugs, such as kanamycin and neomycin.[11] Because premature babies, who are already more susceptible to noise-induced damage, are oftentimes given ototoxic antibiotics to combat sepsis, the levels of noise to which babies are exposed should be of particular concern to health care professionals.

Measurement of Sound

Sound level has both frequency and intensity. Frequency is measured in cycles per second, designated as hertz (Hz). (1 Hz = 60 cycles/s.) The young human ear is sensitive to a frequency range of 20 to 20,000 Hz.[11] Intensity is measured in decibels, a decibel being defined as 10 times the intensity level logarithm and abbreviated by db.[12] Because the human ear is more sensitive to the damaging effects of high-frequency sound (>4,000 Hz) than to low-frequency sound, a better correlation with noise-induced hearing loss is obtained when low-frequency sound is filtered out. The sound level should therefore be measured by use of the A-weighted scale, which is designed to filter out low-frequency sound and whose measurement is designated as dbA. Thus, it is conventionally agreed that the A-weighted scale yields the most meaningful single-number estimate of the probable effect of steady-state sound on human ears.[12]

What sound level is safe for full term newborn babies is not known; moreover, little is known about the risk to premature babies of noise-produced injuries. The Subcommittee on Noise of the Committee on Hearing has shown that continuous noise, like that in hospitals, is more damaging than intermittent noise, which provides an opportunity for recovery.[13]

In the absence of clearly defined data on the relationship between sound levels and disorders of hearing, the American Academy of Pediatrics Committee on Environmental Hazards recommends that sound levels in incubators and neonatal intensive care units be reduced, preferably below 58 dbA,[11] the level found to potentiate the ototoxicity of kanamycin in experimental animals.[10] Gadeke *et al*[14] found that noise levels of 75 dbA disturbed the sleep of infants older than 3 weeks, and a level of 70 to 75 dbA for 3 minutes led to the obvious disturbance of awakening in two thirds of the children evaluated. Therefore, the proposed 75-dbA sound-level limit for equipment suggested by the Emergency Care Research Institute seems to be unacceptable,[15] and we health professionals must do our part in developing the means to protect infants from the potentially harmful effects of noise, regardless of the controversy over the maximum sound levels allowable for the neonatal intensive care unit.

The study reported here investigated sound levels produced by various nebulizers and humidifiers used in conjunction with incubators and oxygen hoods in the neonatal intensive care setting.

Materials and Methods

Materials

Sound levels were measured with a Scott Instrument Laboratory Type 452 sound-level meter, using the A-weighted scale.*

The pneumatic nebulizers studied were 7 randomly selected units each of the Aquapak 621, Bard Parker, Foregger, Inspiron 2305, Ohio Deluxe, and Puritan All-Purpose models.

*Manufacturers are identified in the Product Sources section at the end of the text.

The ultrasonic nebulizers studied were 7 units each of the DeVilbiss 65 and the Monaghan 675 models.

The humidifiers studied were 7 randomly selected units each of the Aquapak 600, B & F, Foregger, Hudson, McGaw Prefilled, Mercury, Puritan-Bennett Cascade, and Travenol Prefilled models. One unit of the Conchapak humidifier was also tested.

Auxiliary devices used in the tests were an Air-Shields C-86 Isolette incubator, an Olympic Medical 10-inch-diameter hard-plastic oxygen hood, an Olympic Medical 8-inch-diameter hard-plastic oxygen hood, and a Mistogen 8-inch-diameter, flexible, soft-plastic oxygen hood.

Methods

The nebulizers were studied by setting up each unit with a 60-inch length of large-bore aerosol tubing leading into the Isolette, whose motor was not running. The calibrated sound-level meter was placed in the Isolette. Background noise level was kept at a constant 40 dbA, which we had found by random measurement to be the average noise level in three neonatal intensive care units during 'quiet' conditions. Blennow *et al* had also reported this background noise level.[16] Each nebulizer was pneumatically operated at various oxygen percentage settings, and sound levels generated in the Isolette were measured on the sound-level meter's A-weighted scale.

The Aquapak 621, Bard Parker, and Ohio Deluxe nebulizers were studied while operating at their 40%, 60%, and 100% oxygen settings. The Inspiron 2305 and Puritan All-Purpose nebulizers were studied at their 40%, 70%, and 100% oxygen settings. The Foregger nebulizer was studied at 100% oxygen only, because it does not have an air-entrainment feature. Each pneumatic nebulizer was studied during oxygen inflow rates of 4, 6, 8, 10, 12, and 15 l/min, under full, half-full, and empty water-reservoir conditions.

The DeVilbiss 65 and Monaghan 675 ultrasonic nebulizers were studied while being operated at their #2, #4, #6, #8, and #10 output settings.

All the humidifiers except one were studied in the same fashion as were the nebulizers, but on their 100% oxygen settings and with small-bore tubing. The exception was the Foregger humidifier, which was studied with both small-bore and large-bore tubing.

Statistical Analysis

Sound levels recorded during the study were compared statistically by analyses of variance.

Results

Pneumatic Nebulizers Used with Incubators

The results of testing the pneumatic nebulizers attached to the Isolette are shown in Table 1. Except for the Ohio Deluxe nebulizers, the nebulizers produced statistically significant increases ($P < 0.001$) in dbA as input oxygen flows rose from 4 to 15 l/min, regardless of water levels in their reservoirs. The change from full to empty water reservoirs caused statistically significant increases ($P < 0.05$) in dbA in all nebulizers except the Inspiron model. We also found that sound levels were significantly increased ($P < 0.001$) when air-entrainment devices were operating to provide 40%, 60%, or 70% oxygen.

Under all testing conditions, the mean sound level of the Ohio Deluxe nebulizer exceeded the 58-dbA level recommended as a maximum by the American Academy of Pediatrics Committee on Environmental Hazards.[11]

The Aquapak 621 and the Puritan All-Purpose models produced sound levels above the recommended maximum of 58 dbA at low oxygen percentage settings, at high flowrates, and at lower water levels in reservoirs. The Puritan All-Purpose nebulizer produced sound levels below 58 dbA at the 70% and 100% oxygen settings with full reservoirs at all oxygen flows, and the Aquapak 621 nebulizer performed in the same way at its 60% and 100% oxygen settings.

The Bard Parker, Foregger, and Inspiron nebulizers all produced sound levels below or at 58 dbA except with dry reservoirs, with the exception that the Inspiron device produced sound levels greater than 58 dbA when set for 40% oxygen and with input flows greater than 8 l/min.

Table 1. Sound Levels in dbA of Nebulizers Attached to Incubators under Various Conditions

Nebulizer	FIO_2	dbA Range (4-15 l/min)			$\bar{X}$			ΔdbA from 4-15 l/min		
		Full	Half Full	Dry	Full	Half Full	Dry	Full	Half Full	Dry
Aquapak	0.40	49-60	53-63	53-66	56	60	61	11	10	11
	0.60	47-56	53-62	52-65	54	59	60	9	9	13
	1.00	49-57	56-60	55-69	55	59	65	8	4	14
Bard Parker	0.40	47-58	45-54	46-58	54	51	54	11	9	12
	0.60	45-53	45-52	51-61	50	50	59	8	7	10
	1.00	44-51	47-56	49-60	49	53	58	7	9	11
Foregger	—	—	—	—	—	—	—	—	—	—
	—	—	—	—	—	—	—	—	—	—
	1.00	41-44	41-44	47-60	42	43	55	3	3	13
Inspiron	0.40	46-63	45-63	47-59	56	57	54	17	18	12
	0.70	44-57	43-57	46-58	52	52	53	13	14	12
	1.00	44-56	43-56	46-61	52	51	54	12	13	15
Ohio Deluxe	0.40	62-64	63-63	57-68	64	65	63	2	0	11
	0.60	63-60	60-61	52-68	62	63	67	−3	1	16
	1.00	61-60	59-63	61-67	61	64	63	−1	4	6
Puritan	0.40	51-60	49-51	48-69	56	56	60	9	12	21
	0.70	46-56	49-61	46-66	53	55	57	10	10	20
	1.00	46-55	46-56	46-66	51	52	58	9	10	20

Pneumatic Nebulizers Used with Oxygen Hoods

Table 2 shows the results of testing the pneumatic nebulizers in three different oxygen hoods under full-water conditions and at various flowrates. Sound levels increased significantly ($P < 0.001$) under all conditions. The largest mean increase in dbA occurred when we used the Foregger nebulizer with all three oxygen hoods; the smallest change in sound level, 4 dbA, occurred when the Inspiron nebulizer was used on its 100% oxygen setting with the soft medium-size oxygen hood.

Sound-level measurements did not differ significantly among the three different types of oxygen hoods ($P > 0.05$), except with the Ohio Deluxe nebulizer. With the combination of this nebulizer and the oxygen hoods, significant differences ($P < 0.001$) in sound levels occurred: sound levels with the large hood were significantly lower than those with the soft medium-size hood, and sound levels with the soft medium-size hood were significantly lower than those with the hard medium-size hood.

Ultrasonic Nebulizers

We tested the DeVilbiss Model 65 and the Monaghan Model 675 ultrasonic nebulizers only with the Isolette incubator, not with the oxygen hoods. They were operated at the number 2, 4, 6, 8, and 10 output settings and yielded constant, low sound levels of 44 dbA (DeVilbiss) and 43 dbA (Monaghan) at all output settings.

Humidifiers Used with Incubators

The results from testing the humidifiers with the Isolette incubator are displayed in Table 3. The

Aquapak 600 humidifier could not be tested under full-water conditions because water began to bubble out of the humidifier into the tubing whenever input flows exceeded 6 l/min. This occurred with all 7 Aquapak 600 units. All the humidifiers that we did evaluate showed a significant increase ($P < 0.001$) in dbA whenever input flows were increased from 4 to 15 l/min, regardless of the water level.

However, at any given same liter flow, the humidifiers evaluated showed a decrease in sound level when progressing from full-water to dry conditions, except for the B & F humidifier, whose sound level remained the same when its water level changed from full to dry.

All the humidifiers we tested had sound levels below the recommended maximum of 58 dbA, except the Travenol humidifier at 15 l/min at full and half-full water levels. The lowest sound levels were found with the Bennett Cascade, the Foregger with small-bore tubing, and the Hudson humidifiers: all produced a mean sound level of 43 dbA. One Conchapak unit was also tested and produced low sound levels, from 42 to 44 dbA, with a mean of 43.5 dbA. The humidifiers produced significantly lower sound levels ($P < 0.001$) than did the nebulizers.

Humidifiers Used with Oxygen Hoods

Table 4 shows the results of testing the humidifiers at various flowrates under full water-level conditions with the three different oxygen hoods. We did not test the Aquapak humidifier under these conditions because of the problem encountered when testing this device with the incubator, nor did we test the Hudson humidifier, because of a lack of supplies.

Table 2. Sound Levels of Nebulizers at Various Flowrates, Used with Three Different Oxygen Hoods

		dbA Range (4-15 l/min)			$\bar{X}$			ΔdbA from 4-15 l/min		
Nebulizer	FIO_2	Medium, Hard Hood	Large, Hard Hood	Medium, Soft Hood	Medium, Hard Hood	Large, Hard Hood	Medium, Soft Hood	Medium, Hard Hood	Large, Hard Hood	Medium, Soft Hood
Aquapak	0.40	62-73	62-70	62-73	69	68	69	11	8	11
	0.60	61-68	59-68	62-67	66	65	65	7	9	5
	1.00	70-75	66-69	64-69	73	68	67	5	3	5
Bard Parker	0.40	59-68	56-67	55-67	65	63	63	9	11	12
	0.60	55-62	51-62	53-64	59	58	66	7	11	11
	1.00	56-62	52-60	53-60	60	57	57	6	8	8
Foregger	—	—	—	—	—	—	—	—	—	—
	—	—	—	—	—	—	—	—	—	—
	1.00	62-70	59-68	59-67	67	65	65	8	9	8
Inspiron	0.40	56-71	55-68	53-69	66	64	63	15	13	16
	0.70	52-64	56-63	49-63	60	61	58	12	7	14
	1.00	52-63	56-64	49-60	60	62	56	11	8	13
Ohio Deluxe	0.40	72-72	67-66	69-69	74	68	70	0	−1	0
	0.60	69-72	65-64	70-66	73	64	69	3	−1	−4
	1.00	68-72	63-63	71-66	73	63	69	4	0	−5
Puritan	0.40	59-70	59-68	60-70	66	64	66	11	9	10
	0.70	57-67	53-63	55-66	63	58	61	10	10	11
	1.00	56-66	50-63	54-65	62	57	59	10	10	11

Table 3. Sound Levels of Humidifiers Attached to Incubators, at F_{IO_2} 1.00 and Various Flowrates and Water Levels

Humidifier	dbA Range (4-15 l/min)			$\bar{X}$			ΔdbA from 4-15 l/min		
	Full	Half Full	Dry	Full	Half Full	Dry	Full	Half Full	Dry
Aquapak	—	44-58	40-56	—	50	47	—	14	16
B & F	40-52	40-52	40-52	44	44	44	12	12	12
Bennett Cascade	41-45	41-43	40-46	43	43	41	4	2	6
Foregger (large-bore tubing)	40-54	40-49	40-43	48	44	42	14	9	3
Foregger (small-bore tubing)	40-48	40-48	40-43	43	43	41	8	8	3
Hudson	40-48	40-48	40-44	43	43	41	8	8	4
McGaw	42-45	41-55	40-55	48	47	47	13	14	15
Mercury	40-54	40-52	40-51	46	46	43	14	12	11
Travenol	41-60	41-59	40-52	51	50	45	19	18	12

Except for the Travenol humidifier, we found that sound levels increased under all conditions when they were used with oxygen hoods ($P < 0.001$), the increases ranging from 3 to 17 dbA. The greatest change occurred with the Foregger humidifier with large-bore tubing.

Sound levels did not change significantly when we used the humidifiers with the soft medium-size plastic hood compared to when we used them with the hard-plastic hood. There were no significant differences ($P > 0.05$) in sound levels between use of the humidifiers with the large-size or medium-size hard-plastic oxygen hood, except for the B & F humidifier, which produced lower sound levels with the medium-size hard hood ($P < 0.05$).

When the environmental sound level was 40 dbA, all the humidifiers we tested with the three different oxygen hoods produced sound levels below the recommended maximum of 58 dbA, except the Foregger humidifier with large-bore tubing. We found that the humidifiers in use with the three oxygen hoods produced significantly lower sound levels ($P < 0.001$) than did the nebulizers with the oxygen hoods.

Discussion

Sound-level data provide information that is necessary to rationally select and use equipment in the neonatal intensive care environment. We found that humidifiers produce significantly lower sound levels than do nebulizers, under all our testing conditions. All humidifiers tested produced sound levels below the recommended 58 dbA except the Travenol humidifier at 15 l/min under full and half-full water-level conditions. We therefore recommend that humidifiers be used in preference to nebulizers whenever possible, because they produce less noise, especially when input flows are 8 l/min or less. We recommend using a Cascade humidifier whenever high humidity is required. When humidifiers are used that have connection capabilities for large- and small-bore tubing, we suggest that small-bore tubing be used, because our data indicate that it produced less noise.

When using nebulizers in the neonatal intensive care environment, one should maintain water levels at half full or more at all times, because this is a factor in noise reduction. The Ohio Deluxe nebulizer produced excessive sound levels under all testing conditions; therefore, we would be inclined not to use it in the neonatal intensive care unit. The Puritan All-Purpose nebulizer and the Aquapak nebulizers also produced sound levels in excess of the recommended 58 dbA at the lower water levels, lower oxygen percentage settings, and higher flowrates. Whenever we used the air-entrainment devices, sound levels increased significantly, so we recom-

mend that flowrates be kept at 8 l/min or less to minimize noise. The Bard Parker, Foregger, and Inspiron nebulizers all produced sound levels below 58 dbA and we recommend their use in the neonatal intensive care unit for noise control when a nebulizer is preferred to a humidifier.

Although the DeVilbiss and Monaghan ultrasonic nebulizers produced low sound levels, they also produce high-frequency sound (ultrasound). Broadband noise generation containing high-frequency sound has been demonstrated to be more upsetting than that containing low-frequency sound at the same level.[11,12] Thus, more research is needed to determine the relationship and effects of ultrafrequency sound on the neonate. In addition to dbA measurements it would be desirable to have a broad-spectrum analysis, commonly referred to as octave-band analysis, to compare examples of equipment that produce different sound frequencies.

We found that the use of oxygen hoods increased sound levels significantly under all conditions in both nebulizers and humidifiers, with the one exception of the Travenol humidifier. Further research is needed to determine the effects on sound levels of using different sizes and types of oxygen hoods.

Some of the sound levels recorded in this study were of sufficient intensity to interfere with the sleep of and possibly damage the hearing of neonates receiving aminoglycosidic antibiotics.[2,9] Further study should be done on the susceptibility of infants to noise and on the levels of noise at which damage occurs. An assessment of children with hearing loss indicates that acquisition of the traumatic deafness type of hearing loss is compatible with acoustical trauma and implicates noise levels in the neonatal intensive care unit as the probable etiology.[17-19] Until further studies determine the precise sound levels that are dangerous to the neonate, the 58 dbA maximum limit suggested by the American Academy of Pediatrics should not be exceeded.

Product Sources

Humidifiers

Aquapak 600: Respiratory Care Inc, Arlington Heights IL

B & F: B & F Medical Products Inc, Toledo OH

Conchapak: Respiratory Care Inc, Arlington Heights IL

Foregger: Foregger Medical Div, Puritan-Bennett Corp, Langhorne PA

Hudson: Hudson Oxygen Therapy Sales Co, Temecula CA

McGaw Prefilled: McGaw Respiratory Therapy Products, Div of American Hospital Supply, Irvine CA

Mercury: Mercury Enterprises Inc, St Petersburg FL

Puritan-Bennett Cascade: Puritan-Bennett Corp, Kansas City MO

Table 4. Sound Levels of Humidifiers Used with Oxygen Hoods, at F_{IO_2} 1.00 and Various Flowrates

Humidifier (Full Water Level)	dbA Range (4-15 l/min)			$\bar{X}$			ΔdbA from 4-15 l/min		
	Medium, Hard Hood	Large, Hard Hood	Medium, Soft Hood	Medium, Hard Hood	Large, Hard Hood	Medium, Soft Hood	Medium, Hard Hood	Large, Hard Hood	Medium, Soft Hood
B & F	43-50	45-56	45-53	47	52	49	7	11	8
Bennett Cascade	44-52	46-51	46-54	49	49	51	8	5	8
Foregger (large-bore tubing)	46-79	48-74	49-70	65	63	62	33	26	21
Foregger (small-bore tubing)	45-55	47-57	46-59	50	50	52	10	10	13
McGaw	47-60	44-54	47-60	54	49	53	13	10	13
Mercury	44-62	43-62	47-61	53	52	54	18	19	14
Travenol	45-64	46-50	48-60	55	49	54	19	4	12

Travenol Prefilled: Travenol Laboratories Inc, Deerfield IL

Incubator

Air-Shields C-86 Isolette: Narco Air-Shields, Div of Narco Scientific, Hatboro PA

Nebulizers, pneumatic

Aquapak 621: Respiratory Care Inc, Arlington Heights IL

Bard Parker: Becton Dickinson Respiratory Systems, Lincoln Park NJ

Foregger: Foregger Medical Div, Puritan-Bennett Corp, Langhorne PA

Inspiron 2305: Inspiron, Div of CR Bard, Rancho Cucamonga CA

Ohio Deluxe: Ohio Medical Products, Madison WI

Puritan All-Purpose: Puritan-Bennett Corp, Kansas City MO

Nebulizers, ultrasonic

DeVilbiss 65: DeVilbiss Health Care Div, Somerset PA

Monaghan 675: Monaghan Medical Corp, Plattsburgh NY

Oxygen hoods

Olympic 10-inch hard plastic: Olympic Medical Corp, Seattle WA

Olympic 8-inch hard plastic: Olympic Medical Corp, Seattle WA

Mistogen 8-inch soft plastic: Mistogen Equipment Co, Oakland CA

Sound-level meter

Type 452: Scott Instrument Laboratory, Div of Eastern Air Devices Inc, Cambridge MA

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Efficacy of the Minimal Leak Technique of Cuff Inflation in Maintaining Proper Intracuff Pressures for Patients with Cuffed Artificial Airways

David Off BS RRT, Sheldon R Braun MD, Bonnie Tompkins MD, and George Bush MD

The minimal leak technique (MLT) of cuff inflation protects against high intracuff pressures (IP) in patients intubated with high-pressure, low-volume cuffed tubes. To evaluate MLT for efficacy with high-volume, low-pressure (HVLP) cuffed tubes, 193 measurements of IP were made in 101 intubated patients. IP measurements were divided into Group 1 (≤25 cm H_2O) and Group 2 (>25 cm H_2O). Group 1 consisted of 53.4% of all measurements. The mean IP of 23.1 ± 2.4 cm H_2O suggests that the IPs were in the safe range. Group 2 consisted of 46.6% of the total measurements. Group 2A, 22.3% of the total measurements, had a mean IP of 37.4 ± 10.0 cm H_2O that could not be corrected to a safe range by use of MLT. Group 2B, 24.3% of the total measurements, had a mean IP of 35.2 ± 9.9 cm H_2O that could be corrected to a mean of 23.3 ± 2.2 with MLT, indicating that MLT was not correctly employed initially for this group. The mean static airway pressure of lung and thorax was 20.6 ± 4.3 cm H_2O in Group 1, 30.4 ± 13.0 cm H_2O in Group 2A, and 21.5 ± 5.1 cm H_2O in Group 2B ($P < 0.001$). The use of HVLP cuffs and varying cuff inflation techniques produced safe IPs 53.4% of the time, while the remaining 46.6% of IP measurements were elevated. Use of MLT reduced IP to a safe range for 24% of the initially elevated measurements. Patients requiring higher airway pressures for ventilation may not be protected by the MLT. (Off D, Braun SR, Tompkins B, Bush G. Efficacy of the Minimal Leak Technique of Cuff Inflation in Maintaining Proper Intracuff Pressures for Patients with Cuffed Artificial Airways. Respir Care 1983;28:1115-1120. Key words: Airway management, Artificial airways, Apparatus and instruments, Equipment and supplies, Intubation, intratracheal, Medical devices, Respiratory therapy.)

Mr Off is Respiratory Care Supervisor, University of Wisconsin Hospital and Clinics, Madison, Wisconsin. Dr Braun is Medical Director, Respiratory Care Services, and Assistant Professor of Medicine, University of Wisconsin Medical School, Madison. Dr Tompkins is Associate Professor of Anesthesiology, and Dr Bush is Assistant Professor of Anesthesiology, University of Wisconsin Hospital and Clinics, Madison.

Reprints: David Off BS RRT, Respiratory Care Services, E5/369, University of Wisconsin Hospital and Clinics, 600 Highland Ave, Madison WI 53792.

Introduction

Prolonged intubation of the trachea and positive-pressure ventilation (PPV) with volume-limited ventilators for the treatment of respiratory failure have become commonplace in intensive care units (ICU) over the last 10 years.[1] A cuffed endotracheal or tracheostomy tube is required, especially in adults, to establish and to maintain an adequate airway. The cuff seals the artificial airway within the patient's

trachea, producing a closed system. As well as allowing PPV, a closed system serves to protect the patient's airway from aspiration.[2]

With the use of cuffed airways, several factors can contribute to or potentiate tracheal complications. These include (1) increased lateral pressure on the tracheal mucosa, (2) infection, (3) tube movement within the airway, (4) prolonged intubation, (5) systemic hypotension and resultant hypoperfusion of the trachea, and (6) toxicity of materials used to make or to sterilize artificial airways.[3] Such complications as tracheoesophageal fistula, tracheostenosis, tracheomalacia, and tracheoarterial fistula can follow prolonged intubation.

Excessive intracuff pressure (IP) used when one attempts to seal the airway has been incriminated as a critical factor directly producing or precipitating major tracheal complications.[4] Ischemia of the tracheal mucosa results from excessive IP exerted on the tracheal wall by the tube cuff. A study by Cross[5] has shown that the tracheal arterial-end capillary pressure is approximately 30 mm Hg (42 cm H_2O). Maintenance of IP at levels less than arterial capillary pressure is believed to be critical for the prevention of tracheal ischemia.[6]

Many devices and procedures have been used to prevent IP from producing tracheal or pulmonary complications. Periodic deflation of high-pressure, low-volume cuffs was ineffective.[7,8] Automatic cuff inflation and deflation in synchrony with PPV[9] was found lacking in regard to protecting the airway from aspiration during the exhalation phase when the cuff was deflated. Prestretched high-pressure, low-volume plastic cuffs[10] allowed IP to be reduced; however, this method also produced asymmetrical cuff shape.[11]

This prestretching method led to the present-day high-volume, low-pressure (HVLP) cuff. Continuously inflated HVLP-cuffed tubes have been used to produce a closed airway system for PPV, but they afford incomplete protection from aspiration. Aspiration may occur through folds normally present in the cuff material when the cuff conforms to tracheal shape and is not fully distended.[12] HVLP-style cuffs have made automatic synchronous inflation equipment unnecessary, because airway pressure is transmitted to IP during PPV.[13] HVLP cuffs can be overinflated, resulting in excessive cuff pressure. Based on their experience, Ching and Nealon[14] identified the critical IP for HVLP cuffs as < 25 cm H_2O, as IP below this level produced less tracheal erosion. HVLP cuffs with IP meters have been used in many institutions to assure safe IP.[2]

Safar[15] and Bendixen et al[16] have suggested that the minimal leak technique (MLT), a method of cuff inflation in which a small leak is allowed to occur at peak airway pressure on inspiration, is the safest approach to cuff inflation for high-pressure, low-volume cuffs. However, our review of the literature provided no satisfactory data to support the assumption that MLT produced safe IP for HVLP cuffs. Our study evaluated whether the use of MLT as a method of HVLP cuff inflation produced IP within the safe range.

Subjects, Methods, and Materials

In mechanically ventilated patients in the ICU, respiratory therapy staff measured the intracuff pressure in cuffs that had been inflated by uncontrolled (ie, unknown) techniques; when the initial intracuff pressure was dangerously high, we reinflated the cuff by means of the MLT and again measured the intracuff pressure. Using the ventilator manometer, therapists also recorded the peak and static, tidal and sigh airway pressures.

Subjects

The subjects were 101 consecutive adult patients mechanically ventilated in the ICU during the period July 24 to October 21, 1981. Of these 101 patients, 98 had endotracheal tubes (Shiley Inc, Irvine CA; National Catheter Co, Argyle NY) and 3 had tracheostomy tubes. All tubes had HVLP cuffs. The patients were intubated by medical personnel. These patients' disorders included status asthmaticus, viral pneumonia, trauma, renal failure, sepsis, and the results of cardiac arrest and coronary artery bypass.

Data Collection

Data were collected soon after mechanical ventilation was begun in the ICU and at intervals not less than 12 hours apart, for as long as the mechanical ventilation continued. During each data-collection

session the therapist first measured and recorded the intracuff pressure as he found it when he visited the patient. If the IP was 25 cm H_2O or less, it was considered a satisfactory pressure and the therapist ended his visit. If the IP was 26 cm H_2O or greater, it was considered an unsafe pressure and the therapist reinflated the cuff, using the MLT, and then measured and recorded the resultant IP. In addition, each time intracuff pressure was measured, peak and static airway pressure measurements were made.

Pressure-Measuring Procedure

To measure pressure, we used the National Catheter aneroid cuff-pressure manometer (National Catheter Co, Argyle NY—Fig. 1). The therapist first prepressurized the manometer to 25 cm H_2O to prevent error that otherwise would result from loss of gas compression in the 'dead space' of the manometer and connecting tubing. The therapist then connected the manometer tubing to the cuff pilot line and recorded the pressure reading on the manometer. Manometers were used exclusively to identify initial IP and IP resulting from the use of MLT, not to establish minimal leak.

The aneroid manometers were calibrated with a water manometer prior to and periodically during the study. After the study they were again checked and found to be accurate to ± 1 cm H_2O over the range of pressure observed during the study.

Peak airway pressure measurements were made by observing the pressure reading on the ventilator manometer at end-inspiration. Static airway pressure measurements were made by occluding the ventilator exhalation port during inspiration and observing on the ventilator manometer the plateau pressure during exhalation.

Cuff Inflation Procedure

To inflate a cuff by MLT, the therapist first deflated the cuff and used a stethoscope to auscultate the air leak around the intratracheal tube at the level of the cuff. Then, during an inspiratory phase, the therapist slowly injected air into the cuff pilot line, stopping inflation when he heard no air leak. He then removed a small volume of air from the cuff, sufficient to create a very small air leak that could be auscultated at end-inspiration.

Data Management

We grouped the resultant IP data into two general categories: In Group 1, IP was less than or equal to 25 cm H_2O; in Group 2, IP was greater than 25 cm H_2O. We further divided Group 2 into IP that was not able to be decreased to less than 25 cm H_2O with the employment of MLT (Group 2A) and IP that could be adjusted to less than 25 cm H_2O by use of MLT (Group 2B).

We determined the mean and standard deviation (SD) for IP and for peak and static airway pressure data in each group. The paired t-test was used to compare IP in Group 2B before and after MLT was applied. We also used unpaired t-tests to compare peak and static airway pressures between Group 2A and Groups 1 and 2B. P-values <0.05 were considered significant.

Results

We obtained 193 cuff pressure measurements from 101 patients. The results of these measurements are shown in Table 1. The mean IP in Group 2B post-MLT was significantly lower ($P < 0.001$) than that before MLT was employed. The mean peak and static airway ventilation pressures of the lung and thorax were significantly lower ($P < 0.001$) in Groups 1 and 2B than in Group 2A.

Fig. 1. Cuff-pressure manometer.

Table 1. Results of 193 Measurements of Intracuff Pressure (IP) in 101 Mechanically Ventilated Patients Whose Cuffs Had Been Inflated by Uncontrolled Techniques

	Group 1 IP ≤ 25 cm H_2O ('safe' range)	Group 2 IP > 25 cm H_2O Group 2A*	 Group 2B†	
Percentage of total measurements in group	53.4%	22.3%	24.3%	
Mean IP ± SD (cm H_2O)	23.1 ± 2.4	37.4 ± 10.0	35.2 ± 9.9 23.3 ± 2.2	(initially) (post-MLT)
Mean peak airway pressure ± SD (cm H_2O)	30.8 ± 6.9	41.6 ± 12.8	31.7 ± 7.1	
Mean static airway pressure ± SD (cm H_2O)	20.6 ± 4.3	30.4 ± 13.0	21.5 ± 5.1	

*IP could not be brought down to safe range by use of MLT.
†IP could be brought down to safe range by use of MLT.

Discussion

Our data show that in a general, broad range of ICU patients, the use of HVLP cuffs and uncontrolled inflation techniques produced safe IP for 53% of the total measurements (Group 1). We believe that HVLP cuffs and the relatively low airway ventilating pressures observed in this group produced safe levels of IP even though inflation techniques varied. Group 2 measurements were of two kinds. We found that IP was not correctable with the application of MLT for 22.3% of the total measurements (Group 2A). We also observed that Group 2A had higher peak and static airway pressures. We concluded that patients whose measurements were in this group most likely had 'stiffer' lungs. While not a measurement of compliance, the difference in static pressure especially does suggest that certain mechanical properties of the lung and thorax were present in Group 2A and not in Groups 1 and 2B. We conclude that higher IPs are needed in the presence of high airway pressure to prevent excessive cuff leak and thereby allow for effective PPV in Group 2A. Group 2B, 24% of the total measurements, had IPs that were correctable by use of MLT. Group 2B represents either patients whose airway pressures dropped between cuff filling and our measuring or patients for whom MLT was not used initially. We think it was the latter, because the application of MLT resulted in a safe IP for this group. We were able to readjust the IP value in Group 2B by use of MLT presumably because the ventilation pressures in this group were lower than those in Group 2A.

One factor of measurement technique that must be noted is that the *actual* IP values were very slightly less than the values measured in Group 1 and slightly greater than the values measured in Group 2. This is the result of our using meters prepressurized to 25 cm H_2O and the subsequent pressure equalization between the cuff system and manometer 'dead space.' This technique was used because meters were not continuously connected to the cuff pilot lines and because it also allowed us to separate the patients into two groups, one below and one above a safe level of IP.

Based on our data, we conclude that devoting extra monitoring equipment and personnel time to routine IP monitoring does not benefit the patient. To date, because of our perception that MLT works for routine monitoring, we measure cuff pressure in only two instances, rather than as a routine procedure for all patients. First, in those patients who are systemically hypotensive, IP problems may arise from reduced tracheal perfusion pressures. An IP of 25 cm H_2O may not be within the safe range for these patients.[2] Ventilation with high airway pressure and variables such as inflation hold and positive end-expiratory pressure (PEEP) may need alteration to ensure that they have no detrimental effects on cardiac output, which would exacerbate hypotension.

Medication to regulate blood pressure also may be indicated. Employment of MLT and the measuring of these patients' IP may be helpful in assuring that the lowest adequate IP is used during the period of hypotension. Second, for those patients with conditions such as ARDS, lung fibrosis, or congestive heart failure, who require high airway ventilating pressures (ie, peak pressures >40 cm H_2O), IP may need to be high so that substantial ventilating volume is not lost as cuff leak. When unavoidable cuff leak does occur, we attempt to compensate for it with an increased tidal volume rather than with excessive IP. Measuring IP in these patients may be beneficial. However, strict adherence to MLT provides some control of IP even if the airway pressures are high. MLT provides the lowest and thus the safest IP able to deliver adequate ventilation. This is true only for HVLP-style cuffs, as their design allows them to transmit ventilation pressure to IP.

Plunkett and Astravas-Perrault suggest that cross-sectional tracheal shape should be considered when one chooses cuffed artificial airways.[17] More precision in artificial airway sizing or alternative cuff shapes that could better conform to the uniquely shaped airways may provide an answer to the problems of increased lateral pressure on tracheal mucosa that are observed with low IP.

Optimal tube size is very important; however, the optimum size may not always be available in an emergency. Cuff pressure can be high if the tube is small relative to the size of the cross-section of the patient's tracheal lumen.[13] The cuff then may be fully inflated and still not be able to seal the airway. Too small a tube with the cuff overinflated to produce a seal can cause an unsafe IP. One must evaluate tube size relative to patient size and sex if improper tube size is suspected. A subsequent change to the optimum size tube will depend on the severity of the patient's condition at the time.

One concern in using MLT is the potential for aspiration. Leaks at cuff folds may occur with any use of HVLP cuffs. Using the minimum occlusion volume with an HVLP cuff does not increase the incidence of aspiration.[13] To date, complete protection from aspiration requires a system with inherently high lateral pressures on the tracheal wall in addition to other safeguards.[18] In high-risk situations for patients who must perform an inspiratory-force maneuver or other maneuvers that create negative pressure within the thorax, the cuff should be inflated to the minimum occlusion volume or greater. Once the maneuvers increasing negative pressure within the thorax are completed, the cuff then should be reinflated by MLT.

In conclusion, this study shows that in 193 measurements from 101 ventilated patients, the use of HVLP-style cuffs and uncontrolled inflation techniques produced 103 IP measurements (53% of total) that were within a safe range. An additional 24% of the measurements benefited directly from the employment of MLT, which produced a safe IP. Measurement of IP with cuff pressure manometers may be of benefit when hypotension or high airway pressures are present. These data remind the health care team of the potential problem of tracheal complications, as some patients' IP may have to be high to prevent PPV compromise due to cuff leak. Routine IP measurement for all patients may not be necessary if one employs MLT appropriately, as in almost all cases MLT used with HVLP cuffs will provide the lowest IP for adequate ventilation.

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☐ **Clinical Prediction and Prevention of ARDS (VT20)** — By Leonard D. Hudson, MD. The mortality of patients diagnosed with Adult Respiratory Distress Syndrome has remained unchanged since the term was first coined in 1976. This videotape investigates a system of dealing with potential ARDS patients, which may decrease mortality. The discussion centers on the identification of patients at risk of developing ARDS, their risk factors, and risk criteria. An approach to detect and prevent ARDS is presented.

☐ **Clinical Use of the Swan-Ganz Catheter (VT22)** — By John Marini, MD. Clinical applications of data obtained by Swan-Ganz catheter (SGC) placement and the situations in which SGC placement are useful are described in this video presentation. The clinical value of the clinical variables monitored by SGC and the complications of its placement are detailed, including "damping" of the waveform, "overwedging," and optimal lung zone placement.

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Dedication

It is appropriate on the final pages of this 35th Anniversary Issue to honor the man whose commitment and vision have been in large part responsible for the Journal, its life, its quality, and its integrity—Philip Kittredge. Phil began his association with the Journal and the Association in the early 60s and served as the Journal's Editor from 1969 to 1989, bringing the publication to a preeminent position, respected and recognized by the medical community. During my years with the Journal, he has given me clear guidance and wise counsel based on his long experience. As Adjunct Editor, he continues to serve and to make important contributions. Thank you, Phil.

Pat Brougher

Appreciation of Reviewers

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ADL	— activities of daily living
ARDS	— adult respiratory distress syndrome
C.O.	— cardiac output
CO_2	— carbon dioxide
COPD	— chronic obstructive pulmonary disease
CPAP	— continuous positive airway pressure
CPR	— cardiopulmonary resuscitation
FRC	— functional residual capacity
FEV_1	— forced expiratory volume in 1 second
HFV	— high frequency ventilation
HCFA	— Health Care Financing Agency
HME	— heat and humidity exchanger
ICU	— intensive care unit
IPPB	— intermittent positive-pressure breathing
IPPV	— intermittent positive-pressure ventilation
IRV	— inverse-ratio ventilation
MDI	— metered dose inhaler
MIP	— maximal inspiratory pressure
MMV	— mandatory minute volume
MOP	— mouth occlusion pressure
MV	— mechanical ventilation
P_{CO_2}	— partial pressure of carbon dioxide
PEEP	— positive end-expiratory pressure
PFT	— pulmonary function testing
PSV	— pressure support ventilation
PSV_{max}	— maximum pressure support ventilation
RCP	— respiratory care practitioner
RIP	— respiratory inductive plethysmography
SIMV	— synchronized intermittent mandatory ventilation
T_{TOT}	— transtracheal oxygen therapy
TYRS	— "Test Your Radiologic Skill" feature
$\dot{V}CO_2$	— physiologic carbon dioxide production
WOB	— work of breathing

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Notices

Notices of competitions, scholarships, fellowships, examination dates, new education programs, and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notice to RESPIRATORY CARE Notices Dept, 11030 Ables Lane, Dallas TX 75229.

AARC SUMMER FORUM

The Westin, Vail, Colorado, July 11-13, 1991

AARC ANNUAL CONVENTION SITES & DATES

1990—New Orleans, Louisiana, December 8-11
1991—Atlanta, Georgia, December 7-10
1992—San Antonio, Texas, December 12-15
1993—Nashville, Tennessee, December 11-14
1994—Las Vegas, Nevada, December 12-15
1995—Orlando, Florida, December 2-5

THE NATIONAL BOARD FOR RESPIRATORY CARE

1991 Examination and Fee Schedule

CRTT Examination

EXAMINATION DATE:	MARCH 9, 1991
Applications Accepted Beginning:	November 1, 1990
Application Deadline:	January 1, 1991
EXAMINATION DATE:	JULY 20, 1991
Applications Accepted Beginning:	March 1, 1991
Application Deadline:	May 1, 1991
EXAMINATION DATE:	NOVEMBER 9, 1991
Applications Accepted Beginning:	July 1, 1991
Application Deadline:	September 1, 1991

CPFT Examination

EXAMINATION DATE:	JUNE 1, 1991
Applications Accepted Beginning:	December 1, 1990
Application Deadline:	April 1, 1991

RRT Examination

EXAMINATION DATE:	JUNE 1, 1991
Applications Accepted Beginning:	December 1, 1990
Application Deadline:	February 1, 1991
EXAMINATION DATE:	DECEMBER 7, 1991
Applications Accepted Beginning:	June 1, 1991
Application Deadline:	August 1, 1991

Perinatal/Pediatric Respiratory Care Specialty Examination

EXAMINATION DATE:	MARCH 9, 1991
Applications Accepted Beginning:	July 1, 1990
Application Deadline:	November 1, 1990
Application Fee:	$150

RPFT Examination

EXAMINATION DATE:	DECEMBER 7, 1991
Applications Accepted Beginning:	July 1, 1991
Application Deadline:	September 1, 1991

Fee Schedule

Entry Level CRTT—new applicant:	$ 75.00
Entry Level CRTT—reapplicant:	$ 50.00
RRT Written and Clinical Simulation—new applicant:	$175.00
Written Registry Only new applicant:	$ 75.00
Written Registry Only reapplicant:	$ 50.00
Clinical Simulation Only new and reapplicant:	$100.00
Entry Level CPFT—new applicant:	$100.00
Entry Level CPFT—reapplicant:	$ 80.00
Advanced RPFT—new applicant:	$150.00
Advanced RPFT—reapplicant:	$130.00
CRTT Recredentialing:	$ 25.00
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Clinical Simulation Examination	$ 65.00
CPFT Recredentialing:	$ 25.00
RPFT Recredentialing:	$ 90.00
Membership Renewal CRTT/RRT/CPFT/RPFT	$ 12.00

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Calendar of Events

Not-for-profit organizations are offered a free advertisement of up to eight lines to appear, on a space available basis, in Calendar of Events in RESPIRATORY CARE. Ads for other meetings are priced at $5.50 per line and require an insertion order. Deadline is the 20th of month two months preceding the month you wish the ad to run. Submit copy and insertion orders to: Calendar of Events, RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229.

AARC & AFFILIATES

December 15 in Miami, Florida. The Florida Society for Respiratory Care presents "The Legal Aspects of Medical Litigation," including a formal mock medical malpractice trial, at the Miami Airport Hilton Hotel. Speakers and role-players are all practicing attorneys and/or legal specialists. The due process includes full litigation from deposition to trial. Contact Dave Robbins at (305) 441-6819.

February 21-22 in Kansas City, Missouri. The Kansas and Missouri Respiratory Care Societies, in conjunction with the Respiratory Care Managers Association (KS/MO), sponsor the 3rd Annual Midwest Respiratory Care Symposium at Baptist Medical Center, 6601 Rockhill Rd, Kansas City MO 64131. Contact Clarissa Craig, Program Director, Respiratory Therapy Education, Johnson County Community College, 12345 College at Quivira, Overland Park KS 66210-1299. (913) 469-8500, ext 3939.

February 28-March 3 in Anchorage, Alaska. The Alaska Society for Respiratory Care, the American Lung Association of Alaska, and the American Thoracic Society present their 19th Annual Meeting/Winter Conference at the Clarion Hotel in Anchorage. Contact Walter Hays at the American Lung Association of Alaska, (907) 276-5864.

April 3-5 in Baton Rouge, Louisiana. The Louisiana Society for Respiratory Care presents the 21st Annual Educational Meeting and Exhibition, "Rx for Preservation of the Respiratory Care Profession," at Embassy Suites on I-10. Contact Jim Lanoha at (504) 381-6542.

OTHER MEETINGS

January 13-18 in Miami, Florida. A 6-day/5-night "Cruise for Pulmonary Patients and Friends" departs from the port of Miami to Key West and Mexico, leaving on Sunday and returning on Friday. Group rates include private stateroom (double occupancy required) and all on-board meals and entertainment. Special airfare rates are available upon request. Deadline is December 17. Contact Dave Robbins at (305) 441-6819.

February 10-13 in Sun Valley, Idaho. St Helena Hospital of Deer Park, California, and the American College of Chest Physicians (ACCP) cosponsor a program on "Cardiopulmonary Wellness and Rehabilitation." Contact Darlene Buczak at the ACCP, (708) 698-2200.

February 27-28 in Honolulu, Hawaii. The Kuakini Medical Center and the University of Hawaii-John A Burns School of Medicine sponsor the Asia-Pacific Conference on Pulmonary Rehabilitation and Long-Term Mechanical Ventilation. Contact Kris Hara or Helen Ono at (808) 547-9532.

March 6-8 in Champion, Pennsylvania. The 11th Annual South Central Respiratory Care Symposium will be held at the Seven Springs Mountain Resort. Contact Sandy Smith at (814) 269-2948.

March 10-13 in Denver, Colorado. The National Jewish Center for Immunology and Respiratory Medicine, in conjunction with the American College of Chest Physicians, presents the 3rd International Conference on Pulmonary Rehabilitation and Home Mechanical Ventilation, with concurrent workshops on home ventilator care and pulmonary rehabilitation, at the Denver Hyatt. Contact Adele Gelfand, Conference Coordinator, (303) 398-1359.

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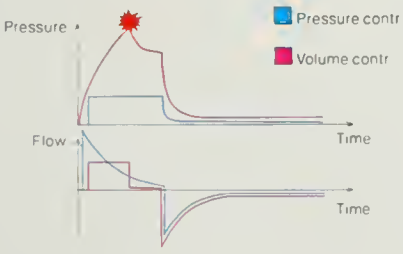

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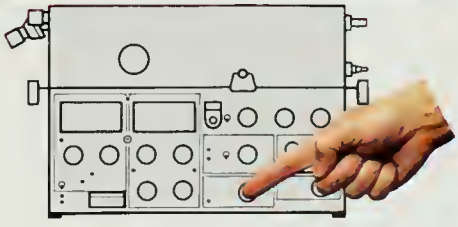

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